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R I C K E T S

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R I C K E T S.

S Y N O N Y M S.

Rachitis; Morbus Anglicus; Articuli Duplicati; Rachitisme ; Nouures; Zwiewuchs; Doppelte Glieder; Rachitismus.

D E F I N I T I O N.

Rickets is a constitutional disease of childhood, characterised by deformities of the skeleton,- such as bending of the diaphyses,swelling of the epiphyses,and the so-called "rickety rosary" or enlargements at the junction of the ribs with their cartilages,- and general impairment of nutrition.

The disease is almost always of long duration, usually with an introductory stage of weeks or months, and a course mostly extending over months or years. Its beginning is mostly gradual,and its final recovery slow. It is complicated with,or dependent on,disorders of the digestive or respiratory apparatuses,which are preceded by a disposition that is probably created by an undue width of the arteries.

It exhibits among its prominent symptoms muscular debility;perspiration;anomalies of the subcutaneous tissue,which is either very much infiltrated with fat or devoid of it;disturbances of the intellectual and moral functions,and of those of the large thoracic and abdominal viscera and lymphatic glands;the changes in the latter perhaps outlasting all others.

The affections of the osseous system,mentioned above,give rise to a peculiar physiognomy,in the absence of which the clinical picture of the disease is not complete or characteristic.

H I S T O R Y.

The independent nature of the disease under consideration dates back less than three hundred years - i. e., to the middle of the seventeenth century, when its prevalence over the whole of England, after having first raged for a time in the counties of Dorset and Somerset, attracted the attention of the Royal College of Physicians of London, who appointed a Commission, of eight members, to investigate and report upon it. This report, prepared by Glisson, Bate, and Regemorter, was published in 1650, under the title: "De Rachitide, Tractatus Opera Primo ac Potissimum Glissonii Conscriptus, Adscitis in Operis Societatem Bate et Regemorter;" Londoni, 1650, and is well worth the perusal of any one interested in the disease.

This is the earliest known publication on the subject of rickets, and the authentic history of the malady does not go back beyond this time. Still, there can be no doubt that the disease had been in existence long before this period, but it had not attracted attention. Stiebel asserts that an ancient statue of Aesop, the fabulist, exhibits deformities which show that he had suffered from rickets in infancy. Hippocrates is supposed to have alluded to rickets in his description of certain deformities (De Articulis); similar allusions have been traced in the works of Galen, Celsus, and Zacutus Lusitanus. Still more distinct is the case described by Baptist Theodosius, of a child seventeen months old, which suffered from extreme weakness, with curvature of the spine and ribs (Epistolae medicinal., 1554). Finally, the statements of various authors of antiquity leave no doubt as to the fact that in the sixteenth century rickets must have been far from uncommon in Switzerland, France, Holland, and Germany (see especially Barthol. Reusner: Dissert. de talo infant., Basil, 1582). Still, it does not seem as though the disease had spread in any exceptional degree over England in the early part of the seventeenth century; at any rate, we know that it became a subject of interest about that time, and was discussed in monographs, among which that of Glisson (Tractat. de rachitide s. morbo puerili, qui "The Rickets" dicitur, Londoni, 1650; ed. ii, 1660; ed. iii, 1671), though subsequent in point of time to those of Whistler (De morbo puerili Anglorum, quem patricio idiomate vocant: "The Rickets", Lugundi Bat. 1645), Garanciers (Flagellum Angliae seu Tabes Anglica, Londoni, 1647), and Boot (Tractatus de affectibus (morbis) omissis, Londoni, 1649), excels its rivals in thoroughness and insight. Glisson himself thought the disease a new one, which - according to the result of enquiries set on foot by himself and his colleagues - made its first appearance in Dorsetshire and Somersetshire between 1612 and 1620. He gave it the name of "rachitis", partly because of the phonetic resemblance of this word to the vulgar English name of "rickets" (from the Anglo-Saxon ricq or rick, a hillock),

partly because it pointed to the special deformity of the vertebral column (ῥάχις). When public attention had once been drawn to the disease,publications about it began toappear in all quarters of Europe,especially in Germany,and discussions set in concerning its true nature and cause. Storch (Theor.und pract.Abhandlungen von der Kinderkrankheiten, Eisenach,1750,iii,S.254) and some other writers contented themselves with the broad explanation that the disease consisted in a disturbance of the growth of the bones and muscles, owing to the unequal distribution of the nutrient juices;others were unwilling to recognise it as an independent disease,preferring to regard it as a symptom of various distinct maladies,chiefly of a dyscrasic order,such a syphilis,scorbutus,scrofulosis, arthritis,etc.Others,again, regarded it as a scrofulosis becoming localised in the osseous system,or as a syphilitic disorder.Finally,the majority followed Glisson in considering it the same as mollites ossium; and among those who warmly advocated this view may be mentioned Trousseau,Lasègue,Stansky,Bouchut,Beylard, and Hohl.Towards the close of the eighteenth century the rise of scientific chemistry,and the progress of normal and pathological histology,shed the first rays of light on the chemical and structural changes that occur in rickets,and on the way in which they differ from mormal processes.The former branch of the subject was investigated by Fourcroy,Bolba,Rees,Schlossberger, Marchand,and Friedleben;the latter chiefly by Rufz, Koelliker,Broca,H.Meyer,Virchow,and H.Mueller.For having conclusively establised the non-identity of rickets with mollites ossium the researches of Virchow deserve special mention.In 1834,Rufz (Gaz.méd.de Paris, 1834) showed that there is a reddish,elastic,reticular tissue in rachitis bones,which Jules Guérin called spongoid tissue.This last-named writer (1837-39) describes three stages of the disease,as follows: (1) Period of incubation or of effusion,in which the blood becomes distributed throughout the interstices of the osseous tissue; (2) period of deformity,in which the spongoid tissue invades the epiphyses,diaphyses,and the subperiosteal space,and the bones soften and bend; (3) period of resorption,of consolidation,and of eburnation,in which the spongoid tissue becomes compact and the bone very hard.To these three periods he later added a fourth,not constantly present,in which the osseous tissue remains fragile and rarefied - the period of consumption.He attempted to make animals rachitic by changing their alimentation,and claimed to have succeeded in so doing in the case of puppies.In 1843, Elsaesser (Der weiche Hinterhopf.Ein Beitrag zur Physiol.und Pathol.der ersten Kindheit.,etc.,Stuttgart and Tubingen, 1843) made another step in advance.He discovered the peculiar change wrought by the rachitic process in the skull,and gave it the name of"craniotabes".Trousseau and Lasègue (Arch.de Méd.,1849) and Beylard (Thèse de Paris,1852) pointed out the resemblance of rickets to osteomalacia. Broca (Société anatomique de Paris,1852)

showed the exact nature of the histological lesions of the disease,and also that rickets,as regards the bones, is only a derivative,an arrest,a suspension of normal osteogenesis.In 1853,Virchow (Arch.f.path.anat.,v., 1853,S.409-507) pointed out its analogies to parenchymatous osteitis,in which view of the nature of the malady both Kassowitz and Baginsky seemed to have concurred. In 1881, Parrot (International Medical Congress of London,1881) claimed to have proved that rickets,both anatomically and clinically is always a consequence of syphilis;but this remarkable opinion, although well presented,has not obtained general acceptance. Other points bearing upon this question will be discussed in due course later on.

M O R B I D A N A T O M Y.

The necessarily limited character of an essay of this kind obviously precludes anything beyond a mere summary of the anatomical alteration in this disease. There is a derangement of all the nutritive processes, which retards and otherwise modifies the growth of the bony skeleton,particularly of the skull,the ends of the ribs,and the ends of the long bones.In cases of rickets that are well-marked, the lesions of the bones are visible to the naked eye,but always on microscopical examination. The long bones are curved or rotated on their axes,sometimes sharply bent at several points;the epiphyses at the wrists and ankles are swollen;the vertebral column may be deviated posteriorly (the condition known as kyphosis) or laterally (scoliosis);there are swellings on each side of the sternum at the junction of the ribs with their cartilages (rickety rosary);the ribs are sunken in laterally,thus narrowing the thoracic cavity and throwing the sternum forward (pigeon breast);and there is bending inwards,etc.,of the flat bones of the skull and pelvis leading to a familiar state of deformity. Trousseau insists on the lightness of the rachitic bones;the entire skeleton of a rachitic child weighed by him did not move the pointer of the machine beyond one kilogramme,instead of eight as would be expected in a child of eight years of age.Even after the morbid process in the bones is concluded,and rarefaction has given place to eburnation,it will be found that the rachitic femur,for instance,weighs less than a normal femur of the same size,and are softer,more spongy,and more friable than usual.

There are various stages in the process of the rachitic alteration of the bones,the same being described by Jules Guérin (Bull de l' Acad.de Méd., 1837,July 13;Gaz.méd.,1839,Nos.28-31) to the following effect:In the first stage,which is that of effusion and rarefaction,the bones are not yet deformed,and are more elastic and less hard than in health;the periosteum is thick,vascular,adherent to the rough and irregular

surface of the subjacent bone.Section of the bone shows that its areolae are dilated,filled by a blood-stained matter resembling gooseberry jam.This jelly-like material is found also in the medullary canal, under the periosteum,and between the separated layers of the diaphysis.This is what is called the medullization of the bones.The cartilage which separates the diaphysis from the epiphysis may be detached by soaking it in water,and is itself thick,bluish,softened,and devoid of the natural firmness.The flat bones present the same areolar condition and the same medullization;the bony tables are thinned,while the diploé is swollen and succulent.The same condition is also present in the flat bones.Decalcification,rarefaction, porosity,and friability are all to be observed in connection with all of the bones when dried.The second stage is that of deformation and organization of the effused material;and in it,if the canges above described continue to increase,the bones yield to muscular action and to pressure,becoming deformed, curved,flattened,twisted,and may be cut into strips. Water injected into the bone will traverse the lamellae and fill all the areolar cavities.It is in this stage that the epiphyses become swollen,that the rachitic bones present the appearance known as the rickety rosary,and that the diaphyses become curved. In the concavity of these curves the periosteum is thicker, more vascular,and more firmly adherent than on the convex side;a new osseous formation separates it from the old bone.The medullary canal has neither the same calibre nor the same extent,nor the same direction;it may be obliterated or it may open externally at the apex of the convexity,when the curve is very pronounced,and one may thus see the two ends of the medullary canal join at an obtuse angle at the apex of the curvature.Usually it has somewhat the form of an hour-glass,owing to its being narrowed in the central portion and dilated at the ends.The spongoid tissue described by Guérin also appears at this stage,and is so called from the fact that the swollen bones suggest a sponge,a biscuit,or gluten bread.One observes a reddish reticular layer,resembling a fine sponge,between the epiphyseal cartilage and the spongy diaphyseal tissue.The fractures,complete or incomplete,sometimes intra-uterine,are due to these lesions.These fractures, ordinarily subperiosteal,unite readily - sometimes with excess of callus;but they may terminate in a pseudo-arthrosis.The long bones are exposed to fracture; the flat bones,especially those of the cranium,present spots of softening,and of spontaneous perforation - the craniotabes of Elsaesser (loc.cit.).This craniotabes,or craniomalacia,affects the occipital and parietal bones,and the petrous portion of the temporal bone,but very rarely the frontal.The lime salts are absorbed,laeving the dura mater and the periosteum almost in contact;the bone is replaced by a soft and depressible fibrous layer,so that a blow or injury may have serious consequences.Now follow the stage of consolidation and eburnation - the third in the

anatomical evolution of the disease.This stage of reparation comes on after the longer or shorter period of disorganization and destruction just described,but the first is not necessarily concluded before the second begins.The new tissue effused beneath the periosteum,between the layers of the diaphysis,in the epiphyseal areolae,becomes organized and calcified. The bones then become harder at the same time that they become straighter,and the nodosities gradually become less marked.Osseous tissue replaces the osteoid tissue described by Virchow (loc.cit.),and the bones becomes harder and denser than normal - i.e.,eburnated.It sometimes happens,however,that this reparation does not take place,the bones remaining spongy and rarefied, and their areolae become filled with fat;they are then fragile,light,and porous.This condition constitutes the stage of consumption.

The osseous lesions of rickets,as observed with the aid of the microscope,were first satisfactorily described by Broca,who takes into account the process of normal ossification for the sake of comparison. The dia-epiphyseal cartilage,which is the active agent in the growth of the long bones,passes,before arriving at the condition of perfect bone,through the intermediate stages called chondroid and spongoid. This layer of pearly hylaine cartilage,which separates the body of the bone from its head,becomes opaque and thicker,proliferates,and becomes transformed into spongy tissue with fine,brittle trabeculae before becoming compact tissue.In rickets the ossification is interfered with;the chondroid and spongoid tissues accumulate at the extremities of the diaphysis without undertoing their final osseous transformation.In the case of the short and the flat bones,no spontaneous tissue is formed,but medullization occurs with softening and decalcification.If,for example,asection be taken vertically through the shaft of such a long bone of the femur in a state of growth - beneath the diaphysis and the intermediary cartilage is seen a bluish layer of one-half to two millimetres in thickness;this is the normal chondroid layer.It is formed of primary cartilaginous capsules,containing secondary capsules in parallel rows,separated from each other by what may be termed rivers,as it were, of a granular substance.This is the "rivulation"of Broca.The lime salts are deposited first in the midst of these tracts or rivers of granular substance,and then in the primary capsules.The secondary capsules are dissolved,the cells which they contain proliferate and fill the primary capsules,which finally form communications with each other,through partial absorption of the calcified partitions which separate them.This is the normal spongoid layer which has succeeded the chondroid layer. The manufacture of bone is now accomplished after the vessels penetrate this layer,and the canaliculi are formed.In rickets,however,this normal process of ossification is interfered with;to the extent that the normal chondroid layer,or layer of proliferated cartilage,increases greatly,attaining a thickness of from four to twelve millimetres instead of one or two.The normal

spongoid layer,or layer of proliferated and calcified cartilage,is also thicker;it is,as it were,mortised with the preceding,and there may even be separate islets of spongoid tissue in the substance of the chondroid material,and vice versa. The vessels are dilated.Beneath this layer appears a red,vascular, spongy tissue having the appearance of bone partially softened with acids.The secondary capsules of the proliferated cartilage are not dissolved,but become calcified;the vessels increase in size,anastomose with each other,and form a cavernous tissue.Under the periosteum the exuded material is transformed into connective tissue,and then into the osteoid tissue of Virchow. When the diaphysis becomes curved,the osteoid tissue accumulates in a thick layer in the concavity of the curve,but remains thin on its convexity.This tissue is also deposited under the periosteum of the flat bones. In the shoft bones we find a no less marked disturbance of ossification;islets of cartilaginous tissue,surrounded by the spongoid substance,are seen here and there, there is obvious rarefaction of the bone,and the vessels of the same are more numerous.In short,the intermediary cartilage which is concerned in the growth of the long bone passes,before becoming perfect bone, through two transitionary stages,- chondroid and spongoid,- which in rickets may become permanent.The chondroid tissue is proliferated cartilage;the spongoid tissue is proliferated and ossified cartilage.Broca also noted,in cases of cured rickets,the persistence of little cartilaginous pearls in the midst of the epiphyseal osseous tissue; the long persistence of which might explain the late deformities,such as genu valgum,coxa vara,the exostoses,and the scoliosis of adolescents.In rickets it is the epiphyses,the growth of which is most active,that present the most marked lesions;the disease,though affecting all the bones at once,does so in an unequal degree.Therefore,in what seems to be partial rickets,linted clinically to a single limb or a single bone,the anatomical alterations are generalized throughout the osseous system.

There is also a considerable alteration of the chemical composition of rachitic bones,the proportions of organic and inorganic constituents being found on analysis to be actually inverted.Normally,the calcareous material in bone represents ttwo-thirds of the substance,the organic matter one-third only;but in rickets the proportin is reversed,the mineral salts constituting not more then one-third of the entire mass.The quantity of lime is decreased threefold or more even.This decalcification is the principal fact brought into prominence by chemical analysis.The same condition exists in even greater intensity in osteomalacia,but here it is complicated with the presence of an excessive amount of fat.In addition to this decalcification one finds in rachitic bones a notable increase in the amount of water,with a slight excess of fat and of carbonic acid. A profound nutritive disturbance is revealed in the exaggerated vascularization,the medullization,and the softening of the bones.

More or less marked lesions of the viscera precede or accompany the anatomical alterations in the bones.The abdomen is of much greater size than normal. This condition,which is habitual,even constant,is not due to a narrowing of the thorax,with consequent depression of the diaphragm,but rather to a dilatation of the stomach and intestines,and,in a measure also, to hypertrophy of the liver and of the spleen.The existence of this gastric dilatation can be verified post-mortem.The lower border of the stomach may be found as low down as the umbilicus,even in very young children.It is seldom that this condition is absent in rickets;and the subjects of that affection not uncommonly have enlargement of the liver and of the spleen. Hypertrophy of the lymphatic glands,hyperglobulia,and leucocytosis may be found in association with it. Lesions,of a congestive and inflammatory nature,may be observed in connection with the organs of respiration. Thus,broncho-pneumonia,pulmonary congestion, and emphysema may be produced by the deformity of the thoracic wall,which also may induce and aggravate bronchitis by interfering with the action of the lungs.The pericardium may show milk spots,and the heart is occasionally compressed and displaced.Rachitis patients usually make a very slow recovery as the muscles may become atrophied from disuse,and almost every organ of the body suffers from the constitutional defect.Even the brain does not escape;for it is usually found at post-mortem examinations not to be well developed,the cerebro-spinal fluid is abundant,and the ventricles sometimes dilated.Nervous disorders are very often observed,such as glottic spasm,convulsions,muscular weakness,and pseudo-paralysis.

E T I O L O G Y.

HEREDITY.

The question as to rickets being hereditary is one about which much discussion has existed.In general, it may be said that hereditary influence may be traced with certainty in many cases of the disease. Many women who were rachitic themselves have been known to have rachitical children.But it has been said that the process runs its full course during infancy,and that therefore a direct inheritance from mother to child is an impossibility.Still,we must not forget that the consecutive conditions of the parents may,or will, influence the general condition of the infant,and result in similar disturbances.No rule,however,exists.Dyscrasic parents may have healthy children,and healthy parents sickly or dyscrasic ones.But the probability is greater that diseased children should come from dyscrasic parents than from healthy ones.Tuberculosis in the parents has frequently been accused of being the cause of rickets in the infant -not directly,but in consequence of general impairment of the tissues;and the same may be said for other diseases.If,as some few claim,rickets were a bacterial affection,the microbe would have to be carried through the placental circulation like syphilis.

Charrin and Gley,who placed small animals under the influence of tuberculosis,diphtheria,and bacillus pyocyaneus,think that they saw rachitical anomalies in the offspring.It is,however,more probable that the circumstances under which the two generations of parents and infants live are the cause of rickets in all. Thoroughly rachitical parents who can raise their infants in favourable surroundings do not transmit their own rickets.General debility,poverty,alcoholism, syphilis of the parents,are predisposing causes. Besides,a debilitated,ill-fed,tubercular,or syphilitic infant is apt to contract rickets.

SYPHILIS.

About the end of the eighteenth century Portal spoke of scrofulous,scorbutic,rheumatic,arthritic, exanthematic,and syphilitic rickets.Particularly has syphilis been accused of being the main cause of rickets by some,and even the only cause by others. Thus it was looked upon by Boerhave.This view was strongly opposed by Van Swieten.In our own time Kassowitz has taken up Boerhave's position,and Parrot still more so - even to the time of his death.As his proof he relied mainly upon the condition of the teeth and bones.But those appearances in the teeth,the thin and ragged edges,the friability and the grooving, either horizontal or vertical,which have been considered characteristic of syphilis by Hutchinson and others,have no such dignity;and,moreover,they are not observed in the temporary teeth at all,but in the permanent only;the rachitical softening of the bones also is not found in syphilis at all.Particularly are there no curvatures in syphilis,and no infractions. It is true that marasmus is found in both rickets and syphilis,but it is met with in all sorts of diseases. The changes in the bones of syphilis are found at birth; in rickets they usually develope in later months.When a baby is syphilitic and rachitic at the same time, the syphilis may last very much longer than the rickets, which meanwhile has healed.The internal organs in rickets do not exhibit any such changes as are known to occur in very many cases of syphilis.No gummata are ever found in rickets,and the interstitial inflammation of the internal organs in syphilis is not met with to the same degree in rickets.What Parrot claimed as a desquamative syphilide of the tongue - that is,red insulated spots,denuded of their epithelium,small in the beginning,later extending backwards and increasing in size - is by no means always syphilitic,but is found in a great many cases where there is no suspicion of syphilis.The identity claimed by Parrot,therfore, does not exist.Cazin and Icovesco (Arch.gén.de Méd., Sept.and Oct.,1887) studied carefully 109 rachitic infants.Inthem syphilis was not more frequent than in the average infants observed.The influence of any,and every,constitutional disease of the parents,or the general condition of the offspring,is liable to cause a disposition to rickets.Hence,syphilis may give rise to the disease under consideration by its general influence upon nutrition;and in this fact lies the key to the connection of great nutritive disorders with each other. Rickets will affect the glands;the caseous and suppurative degeneration of the glands will lead to metastatic

processes,to acute tuberculosis,and to other like disorders.In short,syphilis will undoubtedly change nutrition to such an extent as to result in rickets. Finally,in those countries which are famous for the almost entire absence of rickets (Africa,China,Japan), syphilis is of extremely common occurrence.

GEOGRAPHICAL DISTRIBUTION.

The immunity of tropical countries to rickets is striking,the disease being pre-eminently one of the zone of Central Europe and North America and temperate climes.Speaking of North Greenland and the Faröe Islands, v.Haven (Ugeskrift for Laeger,1882,iii,R.vi,185) and Manicus (Bibl.for Laeger,1824,i,15) mention the rare occurrence of the disease;and in I celand (Finsen: Jagttagelser angaaende Sygdomsforholdene i Isalnd, Kjöbenh.,1874,150)also,and in Scandanavia - i.e., Norway (Faye:Norsk Mag.for Laegevidensk,1862,xvi.,16) and Denmark (Brûniche: Bibl.for Laeger,Oct.,1867,279) - it has a subordinate place in the statistics relating to the earliest years of life.Its principal seats are Germany,Holland,France,Belgium,and Northern Italy; while Southern Italy,the southern provinces of Spain, and still more Turkey and Greece,enjoy a notable immunity from it.The malady appears to be just as common in North America as in the large towns of Europe, though at one time affirmed not to be so (Parry:Amer. Jour.Med.Sci.,Jan.,1872,p.17).So far as South America is concerned, I am not aware that rickets occurs there at all.Analysis of statistics of the various charities dealing with cases of rickets,in children under five years of age,gives the following results:Manchester, in out-patient practice,30.3 per cent. (Ritchie:Med. Times and Gaz.,Jan.,1871,9);at St. Bartholomew's Hospital,during several years, 30,3 per cent.(Gee:The Hospital Reports,1868,iv.,69);Prague,from three years' records of the polyclinic,31.3 per cent. (Ritter v. Rittershain:Die Pathol.und Therapie der Rachitis,Berl., 1863);Copenhagen,from six years' observations at the Children's Hospital,8.4 per cent. (Brûniche:Bibl.for Laeger,Oct.,1867,279); Basel,from records of out-patients during 1876-82,15 per cent. (Hagenbach:Jahresberichten aus dem Kinkerhospital in Basel,1876-82); Dresden,from twenty years' observations at the Childr-en's Hospital,20 per cent. (Küttner:Jour.f.Kinderkr., 1856,iv.,Heft.7,8);Berlin,from ten years'observations among the out-patients of the Charité,25 per cent. (Charité-Annalen,1883,viii.,547);Frankfurt-on-Main, from records of the out-patient department of Christ's Hospital for Children,25 per cent.in 1881,28 per cent. in 1882,27 per cent.in a total of 1000 children treated during 1883 (Lorey:Jahrb.f.Kinderheilkunde,1884, xxii.,52);and in Philadelphia,in out-patient practice, 28 per cent.(Parry:loc.cit.).Barret (Arch.de Méd.nav., August,1878,87) says that he has encountered in Beyrout numerous cases of rickets among the poorer class of children;Pruner (loc.cit.,323),however,remarks that the disease is much less common in Syria and Arabia than in Europe.It appears to be unknown on the Kirghiz Steppes.In India,according to Macnamara (Lectures upon the Diseases of the Bones,London,1881,160) the disease

is exceedingly rare among the native children;and Watson (Med.Times and Gaz.,Jan.,1881,26),and numerous Indian practitioners since his time,confirm this statement.Regarding the East Indies,Waitz (On the Diseases Incident to Children in Hot Climates,Bonn,1843,38) of Java says that he has seen a few cases of the mild form of the disease in children of European parentage, but none in the native children.In Cochin China and China,if it occur at all,it is certainly much rarer than in Europe (Beaufils:Arch.de Méd.nav.,April, 1882,267).It appears to be the rarest thing possible to see a case of rickets in Japan,and I am not aware that it is ever encountered in the Pacific;while it is almost unknown in Madagascar,Mayotte,and the other islands in these waters.Bernal (Arch.de Méd.nav., August,1899) affirms,however,that in the New Hebrides the greater part of the children are either scrofulous or rachitic,but in Fiji it is only seen at very long and rare intervals.In Egypt the disease is rare:Pruner (loc.cit.) has seen it only among the upper classes, and in the children of mixed race.Among the Kabyles of Algiers (Bazille&Gaz.méd.de l'Algérie,1868,30), and in the native children of Senegambia (Chassaniol: Arch.de Méd.nav.,May,1865,508;Borius:Ibid.,May,1882, 375),it is very seldom seen;and the same remark applies to the tropical regions of the African continent. It is practically unknown in Madagascar and the Comoro Islands,and it is very seldom that it is seen in South Africa.The same is true for the whole of South America,and the islands off the coast.

RACE.

Rickets is very common among the children of the coloured people,and this may be due to the way such persons suffer from their sojourn in comparatively cold localities.Thus,the negroes resident in America frequently show rachitic deformities,but never when examined in Africa,in which latter habitat the constant exposure to air and sun appears to protect them. The Italians are also said to suffer inordinately.

SEX.

This appears to be without iinfluence as about as many males are affected as females.

AGE.

The appearance of rickets is most often noted from the sixth to the thirtieth month of life,or, roughly ,during the period of the first dentition. It then becomes gradually less frequent till the fifth year of life;and it is very seldom seen from that age to puberty.The statements as to the age at which rickets is most often found,however,exhibit considerable variability;and the reason for this is to be found in the fact that,as a rule,rachitical infants are presented for observation when one or more symptoms are quite prominent.There are those who take it for granted that all cases (Unruh),others that from 11.5(Quisiong) to 80.6 per cent.(Schwarz) of all cases,are congenital.They claim the rosary swellings of the casto-cartilaginous junctures,and craniotabes for them at birth.In the next few months

these anomalies are said to develope more and more. If that be so, all the alleged or actual causes of rickets, such as foul air, improper food, could not exist unless as concomitants. Von Rittershain coincides partly with the above statements. Still, he asserts that many cases date from intra-uterine life. Kassowitz placed the origin of 50 per cent. of rickets in the first two months. There are many foetal cases, particularly among stillbirths. It is met with very early in intra-uterine life; it is found as a congenital affection, continuing to develope after birth when it has originated in the latter half of foetal existence; there is, thirdly, the rickets of early infancy; and, lastly, that of advanced infancy and childhood. Vierordt extends the time of the occurrrnce of infant rickets from the fourth month to the end of the second year. On the other hand, V. A. Hoffmann declares that the disease is rare in the first half year of life. Pini charges 2974 of his 4176 cases to the second year, but does not take account of either cranial bones or muscles. Of 624 cases of rickets enumerated by A. Baginsky, there were 256 less than a year old, 313 in the second, and 63 in the third year. After this time rickets is rare, as far as the active symptoms of the disease are concerned. But still, a retarded form of rickets (rachitis tardiva) has been described by some authors. It is said to occur about puberty, and to exhibit local changes in the bones of genuine rachitical character, but to be wanting in all the other symptoms required for the diagnosis of general rickets. Such cases have been described by C. Lucas (Lancet, June 9, 1883). He found it complicated, now and then, with albuminuria. The occurrence of the latter at that time of life had been referred to by Moxon (Guy's Hospital Reports, 1878). The principal symptoms described by Lucas are scoliosis, talipes valgus, and genu valgum. The epiphyses were slightly thickened; there were pain in the limbs, langour, and pallor. In some of the cases there were also rachitical deformities dating from infancy. He affirms that the rickets of adolescence exhibits symptoms that are less due to softening of the bones than to relaxation of the ligaments. Chiari has reported a case of rickets of an undoubtedly congenital nature. In it there were only twelve teeth. There were no other alveoli, nor was there any sign of the formation of alveoli in the shape of the jaw, which was of the senile character. Other similar cases are also on record. F. A. Burrall (Trans. New York Pathological Soc., vol. i., p. 81) describes that of a female infant who was cyanosed at birth, and had a small head, and general development in a feeble condition. The respiration was shrill and feeble from birth, as though from congenital laryngismus; in a few days it became hoarse and husky. The autopsy proved the larynx to be normal, with no obstructive growths. The last phalanx of the right finger was missing, and the infant was pigeon-breated. Guéniot, at the meeting of the Société de Chirurgie of Paris (June 27, 1883) presented a newly-born baby with well-pronounced rickets of the extremities which had healed at the time of birth. The bones had recovered

their firmness,and the characteristic deformities remained.At the meeting of December 19th he was able to report that the child had exhibited neither symptoms of rickets nor of syphilis since.In regard to the latter,Guéniot and Fournier made a very rigorous examination of the baby's whole family,but were quite unable to glean any information of syphilis having at any time occurred amongst their number. One of the authors referred to above,namely,Kassowitz states that he has examined many still-born infants,and also children dying at any early age,at the Foundling Hospital of Vienna.In a large majority of the cases he found rachitical changes in the ends of the bones.In many of them who lived several weeks he found rickets developed to such an extent that the presumption of the intra-uterine origin became conclusive.The condition of the maternal blood must here be responsible for the occurrence of the disease,and the foetal and congenital occurrence of rickets,therefore,cannot be gainsaid.Both forms are represented in the literature. Neither requires the presence of rickets in one or both of the parents.But the cause of the intra-uterine disease has not been found.Perhaps a disease of the mother,with considerable nutritive disorders,or a defective placental circulation,may be found responsible,- even a specific micro-organism.The foetal form runs its course long before the normal termination of pregnancy;the congenital may have run its full course at birth or complete it afterwards.The bones are found of characteristic nature,the diaphyses suffering more than the epiphyses;even a rachitical pelvis had been met with by Fischer.Early foetal rickets is probably dependent upon a defective development of the very first cartilaginous deposits and the first osseous nuclei;thus,many of the congenital synostoses find a ready explanation.Besides these,abnormal circulation is accounted for.For periosteal circulation at that early period contracts the foramina carrying the blood-vessles,and,while interfering with the size of the bones,the foramen magnum also.Thus,a certain class of cretinism appears to be due to foetal rickets,mainly of the base of the skull,which results in early ossification of the synchondroses,particularly of the sphenoid bone.By the time of birth this may not have terminated. For,after birth the rachitical process developes further, with the usual well-known phenomena of the condition. In such cases as when the rickets is developed in utero, it may be found at birth to have run its course already (foetal rickets),or it may progress after the child is born (congenital rickets,after Winkler).The latter variety - congenital rickets - likewise includes those cases which exhibit the changes in the bones very soon after birth,during the first few weeks,for in such cases the tendency to the disease,and even its origin, must certainly be referred to the intra-uterine period of life.Although the existence of foetal and congenital rickets cannot be denied (as has been done by Boerhave, Van Swieten,Zenavi,and others),we must be careful how to include in this category many cases originally assigned to it by Glisson,Storch,Morel,Klein,and many other writers down to our own time;for many deformities of the skeleton met with in the foetus and the new-born

infant used to be set down, by the older observers particularly, as due to rickets; whereas in all probably they were due to some other conditions of defective nutrition. The commencement of rickets even after puberty has been reported by several observers, though before the development of the skeleton is complete - i.e., up to eighteen or twenty in the female, and twenty-two to twenty-five in the male sex. Thus, Glisson mentions two cases between sixteen and seventeen; Portal, five cases between fifteen and eighteen. Ollier considers certain curvatures of the spine, developing in subjects not previously rachitic, about the age of seventeen or eighteenmas the expression of a rachitic tendency; and Tripier adds five cases in which swellings on the upper and lower extremities made their appearance in young men. In the absence of microscopical descriptions the above cases must be considered as of a very doubtful rachitic nature. The comparative frequency of rickets at different ages will be seen from the following statistical tabulations:

Guérin's Observations.

Period.	Number of Cases.
Before birth	3
During the first year of life	98
" " 2d " "	176
" " 3d " "	35
" " 4th " "	19
" " 5th " "	10
From the 6th to the 12th year of life ..	5
T o t a l	346

Bruenniche's Observations.

Period.	Number of Cases.
During the first half-year	1
" second "	19
From 1 to 2 years	79
" 2 " 3 "	47
" 3 " 4 "	7
" 4 " 5 "	6
" 5 " 8 "	4
T o t a l	163

Van Rittershain's Observations.

Period.	Number of Cases.
During the first six months of life	91
" second " "	175
From 1 to 2 years	154
" 2 " 3 "	62
" 3 " 4 "	15
" 4 " 5 "	7
" 5 " 9 "	17
	521

Ritchie's Observations.

Period.	Number of Cases.
During the first half-year	7
" second "	65
From 1 to 2 years	109
" 2 " 3 "	25
" 3 " 4 "	9
" 4 " 5 "	4
T o t a l	219

STATION.

Among the important factors charged with the production of rickets are bad air and darkness. Moleschott proved an increase in the elimination of carbonic acid with the amount of light striking the skin and retina. Deep-sea animals, though they belong to the vertebrate class, have a cartilaginous skeleton. After respiratory disorders rickets is often observed. It is true, however, that impeded respiration mau be accompanied, or followed by, all sorts of conctitutional disorders. Animals imprisoned in badly ventilated stables have become rachitic. Soldiers' children, locked up in their bad barracks in the East Indies are subject to the disease (Spencer Watson), though those of the poor outside the barracks are not. Large industrial cities have more rickets than the country. J.P.West, however, denies the absence of rickets from the country districts, and refers to the fact that mild cases are but rarely mentioned at all (Univ.Med.Mag., Oct., 1895). In every village in his neighbourhood he found signs of rickets evident. In nine counties surrounding his residence there were, to his knowledge, eight rachitical dwarfs, three of whom were members of well-to-do Scotch families. The working-class factory population is mainly affected, as Jonkownski, from an examination of 3000 poor children in St. Petersburg, has shown. Others have reported to the same effect. Still, the well-to-do are by no means immune; for, like scurvy, rickets may be found in the families of the wealthy under perfect hygienic conditions (Osler). Thus, in Riga the well-to-do suffer like the poor; 86 per cent. of the infants became rachitical because of the long duration of the winter (eight months), during which the infants are kept in the house. When kept in the open air, even the badly fed will thrive. In this country most of the cases of rickets appear to be observed during the spring, the babies having been housed all winter; towards the end of the year, even after the prevalence of summer diarrhoea, there would seem to be less cases.

DIET.

Rickets is largely due to unsuitable or insufficient food; and the importance of this excitant was known, in a general way to even Glisson and his contemporaries. Subsequently, however, J.L.Petit's view became popular, and it was thought that early weaning formed the principal cause of rickets. Petit thought that to prevent rickets it was necessary to keep the infant at the breast till its dentition was complete. Zeviani, however, was not without adherents in the belief that rickets was brought on by precisely the opposite error

- viz.,by keeping the child at the breast for too long a time.Both views are to a certain extent legitimate. The evil does no lie in the mother's milk (supposing its composition to be normal),but in thefact that when the infant is prematurely weaned the nourishment substituted for the mother's milk is unsuited to its digestive powers and the requirements of its organisation;while,on the other hand,when it is kept too long at the breast to milk alone becomes insufficient for its nourishment.At the present time it seems more necessary to insist upon the latter then the former of these considerations;for the agitation which has been carried on, and rightly carried on,by physicians against premature weaning and artificial feeding of infants,has at times led to an error of the opposite kind in the minds of a large number of the oublic.They have been led to believe that an infant cannot be restricted too long to the milk of its mother or of a wet-nurse.Indeed,in the country districts a large - perhaps the larger - proportion of all cases of rickets is furnished by the children who have been kept at the breast until they are a year old,or even older,without being supplied with any additional nourishment,or with very little of it,at any rate.Among the poorer classes the chief reasons why infants are kept so long at the breast are: first,the convenience and cheapness of this mode of feeding them;secondly,the prevalent belief that lactation protects the mother from the risk of renewed pregnancy.Among the wealthier classes there is the added dread of the child's becoming restless;when a wet-nurse is kept,her personal interest tells the same way,and she usually takes good care that the child is not habituated to the use of any other kind of food.It is important to note that it is not the particular kind of food,nor any special quality or constituent of it, that gives rise to the development of rickets.Any diet unsuited to the child's age and the state of its development will bring on rickets,as is proved by the occurrence of the malady in children fed in the most divers ways.Attempts have been made to prove experimentally the power of unsuitable nourishment to generate the disease in the growing organism.Guérin took puppies away from their dam and kept them on a meat diet.They are said to have shown all the signs of rickets,in its most typical form,after four or five months of this treatment,while other puppies of the same litter,suckled in the usual way,remained in good health.The experiments conducted by L.Tripier on cats,dogs,and chickens have proved that while such unsuitable feeding may undoubtedly cause the death of any of the animals subjected to it,it fails to induce true rickets.Others,again,setting out from the fact that the bones of rickety children are less rich in mineral constituents - especially lime and phosphoric acid - than normal bones,have tried to lay blame on the want of those constituents in the food supplied.They refer to the experiemnts of Letellier, von Bibra,Chossat,and Milne-Edwards,junior;Chossat having produced deformity of the bones in pigeons,and Milne-Edwards in dogs,by feeding them on a diet deficient in phosphoric acid and the salts of lime.Here again, as Friedleben proved by repeating Chossat's experiments

and making a careful analysis of the bones, true rickets is not produced; there is merely a diminution in the earthy constituents of the bone-tissue, and the resulting fragility of the bone is entirely passive, and due to atrophy. Moreover, it is very doubtful if we can ever reduce the proportion of lime and phosphoric acid in the bones, even of rapidly growing animals, by withdrawing these compounds from the food; for the accurate analyses of Weiske and Wildt have proved that, even under such conditions, the bones may retain their normal chemical composition. Even the experience of veterinary surgeons and agriculturists, that young animals pastured on soils that are poor in lime are liable to suffer from rickets, does not go for much; for the consentaneous operation of other causes is not excluded, and there exist counter-observations to prove that, even in the absence of any deficiency in the quantity of lime-salts in the food, rickets may be developed. In itself, then, a deficient supply, or a deficient absorption of lime and phosphoric acid, is not capable of producing the rachitical condition. Besides, cow's milk and farinaceous substances, the common substitutes for mother's milk, are not by any means deficient in lime and phosphoric acid. The causes of rickets must be looked for elsewhere, and we must acknowledge that a deficiency of lime in the bones is not the promary factor in the disease. Minute anatomy has long since shown that the essence of the rachitic process does not consist in a mere diminution of the earthy salts in the bones, but far more in an irritation of the osteoplastic tissue. Wegner, moreover, has brought experimental evidence to show that true rickets may be artificially produced by the continued administration of very minute doses of phosphorus (which exert a specific irritant influence upon the osteoplastic tissue), together with a simultaneous withdrawal of lime from the food. The view, first advanced by Heitzmann, that rickets was produced by a farinaceous diet, and that the active agent was lactic acid, was at one time widely held: the fermentative processes being supposed to be set up by the starch. Still, this does not explain the occurrence of the lesions which I have already described in a preceding chapter, even granting that the lactic acid forms a soluble salt by uniting with the lime of the bone, and so removing it from the system.

MALARIA.

Z. Oppenheimer (Deut. Arch. f. klin. Med., xxx., 1881) has affirmed that the main cause of rickets is malaria; or, rather, rickets is presumed by him to be the form in which malaria makes its appearance in young infants. After disposing of other alleged causes of rickets, none of which is proved to give rise to every case, and referring to the anatomical belief that the peculiar hyperaemia and inflammation of rachitical bones is created by the embryonic condition of the growing osseous tissue, he points to the prodromata, amongst which he emphasizes chronic diarrhoea and the nocturnal crying. The latter with its perspiration and subsequent sleep, he claims as evidence of malaria, and as a substitute for the intermittent neuralgia of adults, the

more so as he believes he finds the spleen tumefied. The persistent diarrhoea of these infants is said to be paroxysmal - to take place in the morning, contrary to what is seen in the usual form of intestinal catarrh; the discharges are said to be serous, not tinged with bile; the appetite to be good during the rest of the day; the weight of the body not to be lessened, but anaemia to develope gradually, and fever to occur occasionally. In other cases infants have cold hands and feet, and blue lips, towards evening; the skin is pale, the spleen enlarged; otherwise there are perhaps no symptoms, but the infants try to get uncovered, and have an increase of temperature of from 1° to 3° F., and a perspiring head in the morning. After a while, the rachitical symptoms belonging to the bones and the general system become apparent. After all of the author's ingenious and emphatic assertions and deductions, it becomes evident that malaria - in the severe forms in which it has been found by Arnstein, Rrowicz, Henck, and others to cause bone diseases - may give rise to rickets; but it is also clear that he tries to prove too much. His views are now mainly of historical interest.

BACTERIOLOGY.

That rickets is really due to a specific infective organism has been often suggested, but not yet conclusively proved. The supposed syphilitic nature of the disease had already been mentioned. In 1860, Friedleben claimed an intestinal ferment to be an important etiological factor; and, as stated, Oppenheimer asserted the same for malaria. Kassowitz believed in the activity of microbes; Volland, in that of a specific virus; Chaumier (Med. Infantile, 1894) in epidemic contagious germs that remained in dwellings, for the production of rickets. Hagenbach-Burckhardt (Berl. klin. Woch., 1895) refers to the facility with which tuberculosis and syphilis locate in the bones and to the swelling of the spleen so common in all infectious diseases, as also in rickets. Mircoli (Gaz. d. Osped., 1891) found in the bones of two rachitical children the staphylococcus pyogenes, and, in four cases, the same and the streptococcus, without other pathological lesions: he therefore concludes that they are the causes of the diseases. In the light of other experience this conclusion appears to me to be premature. Marfan and Marot (Rev. mens., 1893) found the bacterium coli commune and the streptococcus in the blood during enteritis; Czerny and Moser (Jahr. f. Kinderh., vol. xxxviii.), under similar circumstances, the staphylococcus pyogenes aureus and albus, the bacterium coli commune, and the bacillus lactis aërogenes; and Smaniotto Ettore (Rev. mens., March, 1897) the bacillus coli commune, and other microbes, in septicaemia; the latter were found in the bones also. Indeed, the intense hyperaemia of the epiphyses, and of the adjoining cartilage, explains why microbes may be found in these tissues, though they need not be anything but accidental. Nor are the experiemts of Charrin and Gley more conclusive. On February 22nd, 1896, they presented before the Biological Society of Paris rabbits, which had become rachitical after the parents had been fed on

toxines of diphtheria and bacillus pyocyaneus,but were not prepared to claim a direct connection with rickets - perhaps an indirect one with enteritis only.Lange (Verhandl.d.12,Vers.d.Gesellsch.f.Kinderh.im Lübeck, 1895,p.144) suggests that some infecting agent may be attached to certain regions and climates,similar to cretinism (which is mostly - by no means always - found in circumscribed territories with mineralogical anomalies),because of the fact that rickets is rare in Iceland,Finland,Northern China,and Japan,and Northeastern Siberia,where the dwellings are very inferior,and the general circumstances of living by no means favourable.

S Y M P T O M A T O L O G Y.

CLINICAL COURSE.

The onset of rickets is very slow.For the reason that its earliest manifestations are not characteristic, it is difficult to fix the precise time of its commencement.These insidious signs of invasion of the disease are usually connected with the digestive apparatus: irregularity of the bowels,frequent diarrhoea,acid vomiting,tympnaitis,etc.The child is restless,pale,and sickly;it often shows unmistakable signs of wasting, and cries when its limbs are firmly grasped,or when it is raised by putting the hands under its arm-pits. Bronchial catarrh is a frequent complication;febrile symptoms may set in,as restlessness and heat of the skin towards evening,and through the night,subsiding towards morning with an outbreak of perspiration.Even without much sign of fever,the child is often bathed in perspiration - especially about the head and the upper part of the chest.When no cause can be made out for these symptoms,and they coexist with the digestive disorderers alluded to above,they may be allowed to stand for indications of commencing rickets.It is not till they have lasted for some time that the characteristic irregularities in the growth of the bones make their appearance;sometimes,though very rarely,the deformities in the skeleton may show themselves in children otherwise well nourished,during the second or third year of life,without any such prodromata;and, indeed,without any appreciable disturbance of the general health.Moreover,similar deformities have,in a few instances,been observed to appear quite suddenly;and the term "acute rickets" has been employed to designate such cases.Any,or every,bone in the body may show the rachitical changes;but the resulting deformities and functional troubles vary according to the patient's age,and according to the degree of development previously attained by his skeleton.The articular ends of the long bones swell,and protude under the soft parts that cover them - more especially in the hands,feet,elbows, and knees.The prominence of the contiguous epiphyses,

between which the articular cavity forms a depression, has given rise to the term "articuli duplicati,for which Zwiewuchs is responsible.In the skull the disease shows itself by delaying the ossification and the closure of the fontanelles and sutureslthe bones themselves, particularly the occipital bone,are soft and flexible in parts,where they may often be depressed by the finger as though they were made of parchment..In conformity with the age of the child,teething may either be checked and proceed in a slow and irregular way,or the teeth which have already cut the gum are arrested in their growth,become discoloured and brittle,and finally drop out.The force applied to the softened bones by the muscles attached to them,and by the weight of the body,leads to further deformity of the trunk and limbs,mostly by causing green-stick fracture of the long bones.The legs usually exhibit an outward curve, and become scimitar-shaped;the fore-arms are bent ina much slighter degree towards their flexor aspect.The thorax is characteristically distorted.The junction of the ribs with their cartilages become thickened and nodular;they may be seen and felt on the side of the chest,like a chaplet of beads curved with its concavity outwards the so-called "rickety rosary" The ribs are laterally compressed en masse,making the sternum project like the keel of a boat.The sides of the chest are sucked in with every inspiration,especially when,as often happens,there is a catarrh of the air-passages; the upper part of the abdomen (epigastric region) is simultaneously protruded,and when the inspiratory act is near its completion,a deep grove (Harrison's groove) makes its appearance between it and the thorax during every act of taking in of the breath - a groove with which the line of the diaphragm corresponds.The subjects of rickets often show spinal curvature.It usually begins as a kyphosis,which may subsequently become complicated with scoliosis.Finally,the pelvic bones may undergo deformity,the sacrum being approximated by those of the pubes.As a rule,the increase in length of the bodyas a whole is delayed.The child cannot be taught to stand or walk alone;or,if already able to walk,it loses the faculty of doing it.It is unwilling,particularly at the outset of the disease,to put its feet to the ground - perhaps from dread of pain,perhaps from a sense of weakness.When the child does attempt to walk,its gait is tottering and awkward,and it is soon weary.In marked contrast to the delayed growth of the trunk and limbs, the hairy part of the head,whose growth is unchecked, seems unduly big,and often sinks down between the shoulders;the abdomen,already swollen by the gastro-intestinal mischief,seems even more prominent in relation to the shrunken thorax. In advanced cases of the disease the child looks like a dwarf;its mind is often well-developed,or even precocious:this is especially the case in older children,in whom the digestive disturbances are less severe.Younger ones are usually best satisfied when allowed to lie motionaless in bed,or to sit on their nurse's arm,and are generally cross and sluggish. It is by no means always that one finds the disease advanced very far.The structural changes are often limited to a few only of the bones,and stop short of complete softening of their tissues;the pain and

functional disturbance may be insignificant;the morbid process is soon checked,the bones consolidated,trifling deformities set right by renewed activity of growth, digestion and nutrition restored,and the disease brought to a close in a few months,leaving only slight thickening of the bones affected behind.This mild form of the disease usually occurs in children whose digestion has never been greatly disturbed,and whose nutrition has never fallen to a low ebb - i.e.,in older children of the well-to-do class.Even when the disease does actually extend farther in such children,its extension takes place very slowly,with intermissions lasting weeks,or even months;the morbid changes are inconsiderable,and the process terminates at last in recovery,just as in the previous instance,only leaving behind it a somewhat larger numder of more or less striking deformities.On the other hand,however,when the digestive disturbances are very marked and obstinate,and the surrounding circumstances of an unfavourable kind,the morbid changes in the bones are usually more severe,the patient's strength gradually fails,the wasting becomes extreme,and - just as in chronic intestinal catarrh with febrile disturbance and pyrexia - death terminates the scene.Sometimes the brunt of the rachitic dyscrasia appears to fall upn the respiratory organs.The weakly infant,with its diminished power of resistance to cold, suffers greatly from bronchitis,which may assume a serious aspect,owing to the deformity of the chest and spine already referred to,and the resulting interference with the respiratory movements;or the bronchitis may pass into lobular collapse or broncho-pneumonia. Furthermore,as the skull does not afford sufficient protection against injury from without,disease of the brain may be occasioned,as likewise of its membranes, by the softening of the cranial bones.Fever may accompnay any one of the above-mentioned forms of rickets;but the fever seems rather to depend on the patient's general state,upon the complications and sequelae of the disease,than upon the actual changes in the bones.Still,the connection between the bone-mischief and the other morbid conditions is undoubtedly close; for the rate at which the former progresses determines the degree to which the other disorders and the febrile symptoms attain,and vice versâ;so that,in the more acute form of rickets,occurring in children above twelve months old,and almost invariably preceded or accompanied by gastro-intestinal catarrh,it remains a question whether the actual rachitic process may not have some part in the production of the existing febrile manifestations.Indeed, this view is further supported by those very rare cases to which I have already alluded to under the name "acute rickets", by way of distinction from the ordinary more chronic variety of the disease.Feist (Zeit.f.Geburtsk.,v.,p. 101) seems to have been the first to draw attention to this form;Moeller,Hauner (Jahresbericht u.s.w.,in Jour.f.Kinderkrankheiten,1867,xlix.),Bohn,Foerster, Hirschsprung,and others have recorded similar cases. Within a few weeks the epiphyses of all,or most of the, long bones become swollen,there are swellings on the cranial bones likewise,and these phenomena are

accompanied by fever.In several of the recorded cases there was simultaneous swelling of the gums and palate, together with disturbance in the digestive functions. The cases hitherto observed occurred in children between four and sixteen months of age,usually well nourished,and placed under comparatively favourable conditions;occasionally,however,the symptoms broke out in children already much reduced.In the latter set of cases,death followed the development of such complications as pneumonia,but in the former series - a majority of the whole number - the disease ran its course within a period of a few weeks,and ended in complete recovery.A marked activity of growth ensued in several instances.The osseous changes in rickets are not of themselves fatal.At the autopsy of a rachitic subject we almo st always find,besides the changes in the bones,some organic lesions of the viscera to account for death.Apart from intercurrent disorders, which stand in no sort of casual relation to rickets, we commonly find evidence of chronic disease in the digestive apparatus - traces of protracted catarrh of the bowels,swollen mesenteric glands,fatty enlargement of the liver,and hypertrophy of the spleen.

ANALYSIS OF PHENOMENA.

THE OSSEOUS SYSTEM.

The principal causes of the rachitical deformities of the bones are,generally speaking,of two kinds. First,they are the immediate result of the enlargement of the bones by the proliferative processes which I have already described,which is more noticeable during life in the epiphyses than in the diapheses,because the former are less thickly covered by soft parts,and therefore more accessible to inspection and palpation. Secondly,deformity may result from the abnormal softness of the bones,due to the structural and chemical changes that take place in their tissue,and render them less capable of resistance to the mechanical forces brought to bear upon them.Strelzoff gives an additional reason for the impaired resisting power of rachitic bones.He finds in them that the disposition of the osseous trabeculae is an abnormal one.Instead of being arranged concentrically in the long bones,as they ought to be,they are more radially disposed,and this arrangement is not so well arranged to withstand the forces acting on the bone.The principal forces acting upon the bones are the weight of the body,muscular contraction,atmospheric pressure,and the pressure exerted by the growth of an organ in a bony case.It is principally the age at which the disease makes its first appearance that determines the alterations in the skull.There are most marked when it begins - as it generally does - before the closure of the sutures and fontanelles - i.e.,during,or very soon after,the first year of life.In typical cases the head appears unusually large,but its circumference,as proved by the measurements of Ritter v.Rittershain,is not really any greater than in healthy children of the same age; its apparent bulkiness is due to its being out of proport-

ion to the face and the rest of the skeleton,whose growth is delayed.Shaw gives the proportion between the cranium and the face in rickety children as 7 1/13 : 1,while in healthy children it only amounts to 6 : 1. The frontal and occipital bones,with the parietal eminences,are very prominent,and the skull thus acquires a distinctly square shape.The anterior fantanelle, instead of gradually closing at about the fourteenth or fifteenth month,remains open,or may even be enlarged by the expansion of the growing brain.It may continue open till the third year,or even longer,its compressibility varying from time to time in accordance with the fulness of the ventricles and cerebral blood-vessels.Its shape is often altered;instead of its usual four-cornered,or even square outline,it presents an irregular margin studded with little projections. The sagittal,frontal,and coronal sutures may be traced to a variable distance from the anterior fontanelle. Together with the lambdoidal suture,their margins are soft and gaping;they appear thickened and pliable.For a considerable period the posterior and the smaller lateral fontanelles may also remain open.In many cases the occiput is slightly flattened,and the oblique diameters are sometimes notequal,so that one side may appear to be entirely flattened.Particularly in the condition of rachitical softening of the cranial bones,to which Elsaesser gave the name "craniotabes", is this the case.The rachitical skull may present any number of more or less marked perforations,in addition to the six fontanelles,whose patency was attributed to the retarded formation of osseous tissue at the edges of the cranial bones,or to the softening of such as are already calcified.Mostly in the parietal bones,sometimes in the occipital,rarely in the frontal,there are a number of spots,of the diameter of one-half to three-quarters of an inch,mostly with steep margins,transparent,without any osseous tissue left,and giving way under gentle pressure of the finger like a sheet of paper or thin cardboard.In fact,the bone is perforated,and these holes can be felt through the integument.Such perforations are usually quite numerous;from five to twenty or more can be felt,or even counted.They are surrounded by normally hard bone,and therefore can be recognised from the flexible part of the cranium extending alomg the sagittal and lambdoidal sutures.Where these results of rachitical softening,craniotabes,are most prominent - i.e.,on the part on which the infant is mostly reclining,- the bone is flattened,and may remain so for life, though in the majority of cases the asymmetry will disappear.The flattening and perforations result from the same causes - viz.,softening of the bones,and pressure upon the bone between the pillow outside and the brain inside.With it go,hand in hand,thick rachitical deposits under the hyperaemic periosteum of other portions of the skull.Where craniotabes is largely developed on the occipital portion,the frontal and the parietal bones (in their anterior halves)are usually thickened. A cross-section with the knife will reveal a diameter of the new osteoid material between the periosteum and

the bone of one-half to one inch in thickness.It is very hyperaemic - even more so than the bone itself, which,when cut into,exhibits an unusual amount of blood.Sometimes the deposits are still larger,and are apt to change the appearance and weight of the skull considerably after recovery has taken place,and the normal osseous tissue has had its place taken by eburnated and sclerosed material.Several cases of cranial sclerosis due to rickets have been described, by E.Huschke,and others.The author named described the skull of a girl of seventeen years,which weighed 4117 grammes,instead of the normal weight of 600 grammes. The meduallary(Haversian)canaliculi were large,and very numerous on the surface,narrow,and very few,in the interior of the sclerotic bones,and the osseous canaliculi were more spherical and irregular in size and shape. The chemical composition was also abnormal,phosphate of lime being 65.59,carbonate of lime 11.12,sulphate of magnesia 1.14,cartilage and fat (very little),etc.22.15 per cent.No fluorate of lime was found.Most of the bones were exceedingly hard,but fragile when tried in small pieces;very white inside,yellowish on the surface,the latter colour being the remnant of extravasated blood, or other pigmentous matter.Another skull in Huschke's possession,and moderately sclerotic,weighed (lower jaw excluded) 1075 grammes; a third,in the museum of the University of Jena,is that of a young baboon,in whom the sclerotic change was evident in all the bones of the cranial vault.It seems that baboons suffer from rickets very extensively.I.B.Sutton (Trans.Path.Soc., xxxiv.,1883,pp.310,312) describes the condition of two of these animals,one of which was six months,the other one year and six months old,when they died.From his account of the post-mortem findings there can be no doubt that the baboons were rachitical in every part of the osseous system.Huschke reports but ten cases of undoubted cranio-sclerosis - those of Malpighi (1697), Cuvier (1822),Ribalt (1828),J.Forster and Bojanus(1826), Ilg (1822),Kilian (1822),Otto (1822),Vrolik (1848), Albers (1851),Huschke (1858).The disease does not affect the auditory bones,the condyles of the maxilla and occipital bones,nor the styloid process of the temporal bone.It is recognizable in the posterior part of the cranium and base of the skull,but affects mostly the bones of the face,and the frontal,parietal,and cribroid bones.Thus,the disease takes its origin in the anterior part of the skull,particularly in the superior maxilla, and proceeds upwards and backwards,terminating in the base of the skull in the neighbourhood of the infundibulum and appendices.Only two of all the cases were observed during life.In all the disease was traced back to early life.The chemical composition of the bones was greatly changed in all:especially was there an increase in the carbonate of lime.Instead of the normal proportion of earthy to organic material - i.e.,2.1,or 1.5, : 1,it was from 3.5 to 4.4 : 1.There is apt to be much perspiration,particularly of the head,with loss of hair upon the occiput,when the craniotabes makes its appearance between the first and third months of life.The veins are more dilated,the skin thinner and paler than on

the average head,the scalp is very sensitive,the babies crie when laid down,feel better when they are taken up, or when they are lying on their faces.Fisher,of Boston, in 1833,first called attention to the fact that the ear placed over the anterior fantanelle often detects a systolic murmur.This sign has been adopted by many authors as an important diagnostic sign of rickets; but it proves nothing more than that an unossified membrane is better fitted than the cranial bones to transmit the sounds generated in the cerebral vessels (probably in the arteries at the base of the brain) to the ear or stethescope.It can be readily understand that it should appear to be specially associated with rickets,when we remember that the fontanelle is usually of abnormal size in the disease in question,and remains open for a long time.

The most striking feature in connection with the facial bones is the defective development of the jaws. This may be absolute or relative,and depends mostly on a reduction in volume,even while the size of the cranium is normal,or sometimes more than normal.Glisson knew of this,and therefore looked for the cause of rickets in the process of dentition.Now,both maxillae are liable to become rachitical at an early date,as early,indeed,as the bones of the cranium.Rachitical deposits and softening take place in them very generally.The mandible is flattened anteriorly,it loses its rounded outline,is shorter in longitudinal direction, while the rami are thick and clumsy;the whole bone is shorter than usual,and sometimes asymmetrical.Its changed appearance is greatly due to the effect the muscles,with their powerful insertions,produce on the softened bone;mainly the masseter,also the mylohyoid, which draws the lateral portion inwards,and the geniohyoid,which pulls at the central portion.Of the latter, the lower portion is drawn out,the inner,and the alveolar part,inwards.Thus,the teeth,mainly the incisors,of the lower jaw are turned inwards to such an extent that, as those of the upper look outwards,the two rows of teeth do not touch,but cover each other.Besides,the periosteal proliferation around the alveoli is excessive,sometimes so much so as not only to crowd the teeth into irregular positions,but even to absorb and annihilate alveolar processes in the course of the morbid changes.The cases in which the number of teeth are actually diminished by rickets are not at all rare.In the superior maxilla the last-described anomaly is also observed.Periosteal thickening is mainly noticed about the intermaxillary bone - sometimes to such an extent that above and behind it considerable impression takes place.The malar bones become very prominent,and the shape of the superior maxilla is more spherical than normal.

The most common effect of rickets is seen in the retardation of the first dentition.Irregular teething is constantly associated with maxillary rickets,but is also present when the latter is not well,or not at all,marked.As a rule,t he first teeth protude late, about the ninth or tenth or twelfth month.That the first year,and more,should elapse in rickets without

any tooth is common enough in rickets.Cases in which the first teeth do not come before the second year is completed are not very uncommon;in some there are none even when the child is much older.In most cases the retardation of dentition goes hand in hand with very marked retardation in the development of the rest of the bones,and in the closure of the fontanelles of the cranium.But not in every case of rickets is there a retardation in the process of teething.In some a few teeth appear at the regular period (at the completion of the seventh or eighth month),or even at a very early age (in the fourth or fifth month);after which there is an interruption in the protusion of teeth for an indefinite period.Evidently the period in which rickets is developed exerts its influence on the teething process.When it exists at a very early age,it will retard teething until recovery takes place.Still,it is possible that a moderate amount of periosteal and osteal hyperaemia,and over-irritation,matures the teeth abnormally.In all those cases,however,in which rickets does not occur before the second half of the first year,the first teeth will appear at the normal time, and a long period will follow in which no teeth at all will make their appearance.Then,again,when the whole process comes to a stand-still,and recovery takes place with the solidification of the bones,and even eburnation,the teeth will come in rapid succession.Whether they will,as is frequent,decay almost as soon as formed, or whether they will be unusually hard,solid,and yellowish,depends on the stage of the disease in which they make their appearance,and on the complications aggravating the case.Digestive troubles,before and during the course of the disease,are of very grave import in this respect.

Deformities of the thorax are also of common occurrence.The ribs of the convex half are prominent and divergent,those of the concave side flattened and parallel.The two halves of the chest are therefore very unequal indeed.Muscular traction,atmospheric pressure,the elastic traction of the lungs,the presence of pulmonary complications,and the pressure from below on the part of the enlarged viscera of the abdominal cavity,are principally responsibe for the deformity of the thoracic cage.It is very much aggravated by the ribs and sternum.Even without any affection of the vertebral column,they suffer severely from the general affection.The manubrium is thickened and drawn inwards,the ensiform process protuberant,the sternum often swelled and painful to the touch.The ribs are sensitive to the touch on one or both sides.The child cries when taken up,or when fearing to be taken up.The osteo-cartilaginous junctions are thickened, mainly so from the fourth to the eighth ribs.The insertion of the diaphragm becomes soon perceptible by a deep groove around the chest.The anterior portion of the ribs is flattened,posteriorly they are inserted at acute angles.Thus the intra-thoracic space becomes narrow,the sternum,with the costal cartilages,is pressed forward (pigeon breast,pectus carinatum),the thorax is deprived of its elliptical shape and becomes triangular,the dorsal apsect being flattened,and the distance between the vertebral column and the sternum

increased.Below the diaphragmatic groove the thorax expands,the liver and other abdominal organs crowding the ribs outward.All sorts of changes are expereienced by the ribs in these conditions.Parts of them are flattened,parts undergo infraction,parts are even concave; they are bent and twisted,now and then to such an extent as to turn the concave side out,the convex surface in.In addition to all this,the scapula is big and clumsy,and protuberant,the clavicle sometimes covered with callus,considerably bent,and frequently infracted, between the middle and anterior thirds.

The rachitical process also affects the spinal column.In the healthy infant this is straight,but in the rachitic it very soon exhibits a kyphotic deformity.When such a baby of three or six months is sitting up,the middle portion of the back is protuding,as in Pott's disease.In almost every case,however,this kyphosis is but apparent,and the result of muscular debility.In order to arrive at a diagnosis at once,it is sufficient to place the patient on his face and to support the head,and raise the lower extremities and pelvis in the air.If the kyphosis is but functional, the prominence disappears at once.By nothing can the muscular insufficiency of early rickets be demonstrated better than by this experiment,simple as it is.But actual deformity is also found in rickets.It sortens both the vertebrae and intervertebral cartilages,and either their anterior or posterior portion may be irregularly developed,and be too high or too low. Besides,the articulating suffaces are sometimes too convex.Thus,the causes of both kyphosis and scoliosis are amply furnished,and complications of the two are frequent,and the deformities resulting therefrom quite formidableScoliosis has mostly its convexity to the right,with compensation above and below,The spinous processes are very frequently directed to the concavity.The intercostal spaces are very narrow on the left side,because the ribs are bent out,and of less curvature than usual.

In a variety of ways the bones of the pelvis participate in the rachitical deformities.The degree and the manner in which they do this dependpartly on the age at which the disease set in,partly on the existence and degree of other deformities.Accordingly, the deviations from the normal standard of pelvic symmetry are various.We do not always find the peculiar form that used formerly to be described as,par excellence,the "rickety pelvis" (a pelvis narrowed in its antero-posterior diameter,obliquelyelliptical,reniform, etc.).Many of the peculiarities of the rickety pelvis may be observed,as Kehrer points out,even in the foetus, and in infants who have not yet learned to walk;so that they must be set down to muscular contraction (ileopsoas,erector spinae,gluteus medius,etc.).There are other causes,too,which tend to increase the variety of pelvic distortions in rickets:the bones of which the pelvic girdle is made up,and the individual elements of those bones,may be affected in various degrees by the softening process;previous deformities of the trunk or lower extremities may influence the pelvis; finally,the nature of the pelvic distortion will be

influenced by the usual attitude of the child when it becomes affected by rickets - no matter whether it is able to walk,or requires to be carried in the arms,or is recumbent.In childhood none of these deformities give occasion for alarm.Their importance does not become apparent,save in the female sex,as a possible hindrance to parturition.There are several kinds of rickety pelvis,all of which are fully described in works on obstetrics.

The extremities suffer in various ways,and at a later period than the ribs and cranium.The opinion of Guérin,that the rachitical process begins in the lower extremities and ascends gradually,is erroneous. It cannot even be stated that the lower extremities are affected sooner than the upper.There is no regularity at all;it is not even necessary that all the osseous tissue should be implicated.But this can be taken as a fact,that the hands and the feet,and particularly the phalanges,are the latest to undergo the rechitical change.First in the line of morbid alteration of the bones are the epiphyses,mainly of the tibia,fibula, radius,and ulna.Their integument appears to be thin; now and then the cutaneous veins are dilated.The periosteum of the diaphyses becomes thick,softened,and painful to touch and pressure,its compact layer thin, the meduallary space large,the whole bone flexible,at the same time that the ligamentous apparatus of the joint becomes softened and flabby.At this time babies appear tohave a marked facility in introducing their feet into their mouths. For,at the same time,the bones begin to curve under the influence of the flexor muscles,which are always stronger,as they do in the later months,under the weight of the body,when the child begins to walk.The curvature is not always a mere arching,but sometimes the result of infraction (green-stick fracture),a complete fracture not being accomplished because both of the thickened and softened periosteum.Both the legs and forearm bend on the external side,the resulting concavity looking inward. The thigh usually bends forwards and outwards;the humerus in a direction opposite to that of the forearm.Curious anomalies are often caused by attempts at walking;creeping,sliding,walking,turn the extremities in such unexpected directions that talipes valgus, genu valgum,and,now and then,double curvatures,are the results.These,however,may not always be very marked, but there is one change in the rachitical bone which is very constant - viz.,the impairment of longitudinal growth.In every case the diaphyses remain abnormally short,and the proportion of the several parts of the body are thereby disturbed.Chiari measured parts of the skeleton of a rachitical woman,of twenty-six years, who was nine years old before she could walk.Her height was116 centimetres,the length of the lower extremities 42,femur 23,tibia 15,fibula 20,humerus 16,right radius 12.5,left radius 11,right ulna 15,left ulna 14 centimetres.In a second case the parts of the skeleton were measured after they had been extended with great care. The right arm,from the acromion to the middle finger, inclusive,was 39 centimetres,the left 38;the right lower

extremity,from the trochanter to the great toe,inclusive,39,the left 41 centimetres.Guérin ventures on a generalisation as regardsthe comparative frequency of the different kinds of deformity,and the order in which they are usually developed.He believes that the lower epiphyses of the bones of the leg are always the first to be affected,the disease then extending upward.This is true only of those cases in which rickets comes on almost imperceptibly,in children upwards of a year old,whose cranial bones are already consolidated.It is not true of that far larger group of cases in which the disease sets in about the time of the first dentition.Hence,the skull is the first part of the skeleton that suffers;its fontanelles and sutures remain open,or may even increase in size;the appearance of the teeth is checked.It is not till later that the ends of the ribs and the epiphyses of the long bones begin to swell;this change showing itself first either in the hands or in the feet,and spreading from themce to other joints.The vertebral column does not appear to be affected till a relatively late stage in the course of the disease.Shaw affirms that the general increase of the stature is checked far more in the lower than in the upper half of the body;the former being about one-third,the latter only one-thirteenth,less than it ought to be.Ritter von Rittershain found that,of forty-two rickety children,one only attained the normal average stature of a healthy child at the same age;all the rest fell short of it by from 3 to 6 centimetres,or even more.Rickety subjects are often stunted for life, even though they may have escaped any of the more serious deformities of the spine or limbs;for it is not until the disease has entirely spent itself that the growth of the body in length is actively resumed;and this resumption of activity is often insufficient to make up for the effects of previous delay.

NERVOUS SYSTEM.

A complication of rickets that is by no means infrequent in its occurrence is hydrocephalus.It is due to the sluggishness of the arterial circulation, and to the presence of intense hyperaemia in the cranial bones and in the meninges.The former is due to the relatively large size of the arteries,and to the feebleness of the muscular contraction all over the body;which,therefore,is but an insufficient excitant of the immense peripheral circulation.In craniotabes there is intense hyperaemia,both in the bones and in the meninges of the brain.Effusions,therefore,are frequent.Small ones are easily absorbed;not infrequently will their absorption,after recovery,give rise to renewed growth of the brain.Like the bones,which,after recovery,are more solid and stronger than normal ones, the brains of rachitical children appear to developevery favourably;the large square heads,characterized by their combination with short and thick-set limbs,often belong to the best scholars at school,and to the most enduring intellectual and physical workers amongst adults.Thus,to a certain extent,the intense hyperaemia of rickets,in some instances,is rather propitious than otherwise.But,if this hyperaemia lasts long and is excessive,so as to disturb nutrition permanently,the resulting effusion will persist and,to a greater or

less degree,actual hydrocephalus follow.In the vast majority of instances genuine hydrocephalus and rachitical hydrocephalus may be differentiated from one another.In some cases this is difficult,for the patent sutures and fontanelles will be met with in both forms, as also,sometimes,in the presence of tumours.It is easy when there is a history of previous rickets.The deposits on the forehead,the presence sometimes of characteristic cranial defects,with steep edges in the middle of the bones,and of laryngismus stridulous,which is rare in genuine hydrocephalus,the condition of the thickened epiphyses,and the curvature of the diaphyses, are our chief guides.During the first stages of rachitical hydrocephalus,the intellect is not disturbed.The hydrocephalic head of rickets is rather spherical; that of genuine hydrocephalus,when external,is often so bulging laterally that it appears flat on the top;when external bulging upward,and even conical and pointed. In the latter two forms the face is comparatively small at an early period,and the intellect impaired.Hebetude, idiocy,paralysis are more easily found in the genuine form,though they be not absent in some rachitical cases. In the former the orbit is soon depressed,and ocular symptoms are more prominent.Ckoked disc,and other pressure symptoms,are met early.The fontanelle pulse and the systolic murmur disappear soon,while in rachitical hydrocephalus they are apt to remain a long time.Still, there are cases in which the diagnosis may become very difficult indeed.It is desirable to come,however,to a conclusion very soon,for a rachitical hydrocephalus is by no means unpromising.Many cases get altogether well, and proportionately very few carry their calamity into the second decade.In many the combination of phosphorus with judicious punctures proves efficient.Lumbar puncture as a means of diagnosis does not answer readily,for the strength of the current through the exploring needle does not correspond with the amount of fluid (which is liable to be less in rachitical than in genuine hydrocephalus) contained in the cerebral cavity.The laryngismus stridulous of infants is one of the common symptoms of the cerebral changes which take place during,and in consequence of,craniotabes.The crowing inspiration in question may be mild or severe.The mild form is very frequent,and comsists in the occurrence of a shrill inspiratory sound while the baby is either quite placid or excited or crying.It is frequently overlooked entirely,is usually overcome after a number of months,and gives rise to serious trouble in but very few instances. The severe form is of a different nature.While the baby is awake or asleep,without any premonitory symptoms, while playing or crying,placid or excited,all at once respiration will cease.This will take place,usually, after expiration.The limbs are hanging down,as it were, lifeless,the face turns pale,then purple,and slight convulsive twitchings may set in for ten or twenty seconds.There appears to be a complete paralysis,and death from apnoea appears to be imminent.All at once,a long,deep crowing inspiration will be heard,respiration will commence again,and the whole terrible attack is overcome.It may return a number of times every day,or sometimes not for several days,during a period of many

weeks or several months.The attacks which set in after inspiration are apt to be more dangerous,and more so than when before that epoch.In such an one,but also in the other kind,which sets in after the expiratory movement,death may occur suddenly,or the attack may be followed by a convulsion,which may terminate fatally like any other eclamptic seizure.In this manner it is that the majority of cases of rickets perish which terminate fatally during the active progress of the morbid process.In this connection,however,it may be well to add that craniotabes is not the only cause of laryngismus stridulous,particularly when the latter is found in the second year of life,or even later.But almost every case,without any exception,which is observed during the first eight or nine months is due to that very cause;and a good many cases occurring later, when the craniotabic bones have become normal,arise from the effects,either meningeal or encephalic,of the rachitic process.Still,complications of craniotabes, with enlargement of the thymus gland,may occur,and enlargement of the tracheal and bronchial glands are by no means rare.Z.Oppenheimer prefers the name "rachitic asthma" in place of laryngismus,and suggests an explanation of the symptoms from a strictly anatomical point of view.If not correct,it is at all events interesting.He points to the ligament situated between spinae intrajugulares of the temporal and occipital bones,which,as long as it is of normal consistency,separates the jugular vein from the pneumogastric nerve. As it is covered with periosteum and dura,it is apt to ossify,and forms an osseous partition in the foramen jugulare,which participates in all the changes taking place in the periosteum.As this becomes softened and succulent,so will the ligament,either on both sides or either.Its influence on the neighbourhood depends on its size or succulence (as also on the difference in width of the foramen jugulare or lacerum,which correspond to the difference in size of the transverse sinuses).The irritation of the pneumogastric is perhaps easily explained thereby,but in very exceptional cases only the accessory nerve would be affected.As,however,the latter controls the sterno-cleido mastoid muscle and trapezius,and the laryngeal muscles,and is apt to provoke cardiac paralysis during diastole,the occurrence of sudden death would be best accounted for.An occasional actual hyperaemia of the thymus does occur without rickets;but in rickets enlargement of the thymus is not uncommon.Besides,there are those cases in which laryngismus stridulus is observed together with rickets,but the former continues after the latter is cured.Such is the case of Kassowitz,who relieved rickets with phosphorus,but not the accompanying laryngismus stridulus; also one of Canali,who found that the antirachitical treatment did not relieve that same condition of the larynx.In that case it was complicated with, or dependent on,hydrocephalus.But these cases form a small minority only:19 out of 20,probably 49 out of 50,are of rachitical origin.There appears to be a special centre in the frontal lobe which,when irritated,causes bilateral abduction of the vocal cords,with complete obstruction, and another one,in close proximity,which during expiration causes interruption of the respiratory acts. These cases are met with at the age at which cranial rickets is common,mostly before the ninth month.That

is the time in which the growth of the cranium and of the brain is most intense;the physiological development is therefore apt to become,on slight provocation, pathological,and the hyperaemia of the brain and meninges,with its tendency to effusion,predisposes to secondary physical and mental disorders.Rickets is sometimes the cause of tetany,but by no means very often.In this condition the joints of the carpus are flexed.The thumb is turned in,the forefingers are extended in the interphalangeal and flexed in the metacarpo-phalangeal joints.There is talipes equinus, sometimes flexion of the forearmlthe neck is stiff and reclined,the muscles are rigid in the contracted extremities.The attacks are intermittent,seldom last for hours or days;consciousness is intact;the paroxysms last for from a few minutes to a few days,and disappear mostly after a few months.Its nervous symptoms share with rickets this peculiarity,that they do not interfere with the intellect,and have a strong tendency to recover after running a limited,and irregular course. S.Stirling (Arch.f.Kinderh.,vol.xx.,1896,p.1) and others look upon three of the symptoms of tetany as pathognomonic: (1) That which is called after the name of Erb,and consists of increased electrical (more galvanic than Faradic,and more motor than sensory)excitability. (2) Trousseau's symptom;that is,compression of the vasomotor brachial plexus,or pressure in the popliteal space, causes an attack of tetany,either through anaemia produced by local pressure,or,rather,by irritation of nerves. (3) Chvostek's so-called facial symptom;that is, pressure on,or tapping,or sometimes even gentle friction of,the zygomatic arch in front of the ramus of the maxilla produces a spasm of the upper lid and alae nasi.Perhaps too much has been made of the latter symptom,and in many of the recorded cases it appears to have been absent.On the other hand,it may be present sometimes inthe healthy,both young and old,mainly the latter,in epileptic,neurasthenic,hysterical,and chlorotic subjects.If that be so - or,rather,as it is so - tetany and the facial symptom are no longer identical. At all events,those rachitical children who exhibit both Chvostek's and Trousseau's symptoms must not,as it has been so often done,be charged with having had "latent tetany".Indeed,a disease must be very "latent" that has no symptoms except a single one whose significance is very doubtful.The symptoms belong to rickets when present,but not to tetany proper.Indeed,cases of complicated rickets,particularly such as are developed early,and exhibit more or less craniotabes,that yield both Chvostek's and Trousseau's symptoms,are not infrequent.Kassowitz recalls 172 rachitical children suffering from nervous symptoms.In 108there was laryngismus stridulus,in 120, the facial phenomenon of Chvostek,in 41, the pressure phenomenon of Trousseau,in 19 tetany.Th-us rickets,and mainly cranial rickets,may be found complicated with tetany.That is why one of the disorders depending on cranial rickets - viz.,laryngismus stridulus,is occasionally met with,orcomplicated with tetany.Escherich frequently observed the occasional coexistence of tetany with laryngismus stridulus.What he,however,emphasizes solely is that he found the

facial phenomenon in many,and Trousseau's symptoms in some,infants suffering from laryngismus stridulus. This appears to him a sufficient reason for disclaiming the latter as a symptom of rickets,and for acknowledging it as a symptom of tetany.One of the very rare alleged complications of rickets is the so-called "salaam spasm"(spasmus nutans),which malady consists in rhythmical convulsions of the sterno-cleido-mastoid, and of the muscles which rotate the neck.The patient exhibits a nodding and rotatory movement.It is often complicated with nystagmus of one or both eyeballs, and sometimes of the eyelids,also with strabismus,and with petit mal.Convulsions are an uncommon complication.The etiology is not always clear.Falls on the head are not infrequently charged with being the cause. Many cases in which the head and eye movements are combined point to a defective equilibrium in the special cerebral centre.Rickets has often been accused of being the cause,and has just as often been denied. Among the other manifestations of rickets on the nervous system Epstein (Prager med.Woch.,1896,Nos.43, 44) includes kataleptic symptoms.These and other phenomena are,however,quite rare,and in very many cases of rickets are entirely absent.

THE SKIN.

This also participates in the general disorder of nutrition present in rickets. It is soft and flabby. In those infants who becpme rachitical gradually, while proving their malnutrition by the accumulation of large quantities of fat,it exhibits a certain degree of consistency.When rickets developes in the second half of the first year or later,with the general emaciation the skin appears very thin,flabby,unelastic. The veins are generally large.Complications with eczema and impetigo are very frequent;where they are found the glandular swellings of the neck,and below, are still more marked than in uncomplicated cases. Circumscribed alopecia is sometimes found,apart from the extensive baldness of the occiput.It is not attended with,or depending on,the microsporon Audouini,but the result of a tropho-neurosis.In the hair Rindfleisch found fat-globules between its inferior and central third.Then it would break,the axial evolution would cease,and,by a new formation of cells,the end would become bulbous.The copious perspiration of rickets leads to the development of miliaria and other eruptions,such as eczema.Pruritus is sometimes a complications thatgives great annoyance to the patient,and acute lichen is not uncommon.After recovery the skin is apt to be dry and desquamative.

RESPIRATORY SYSTEM.

One of the very frequent complications of rickets is bronchial catarrh.Owing to its being badly nourished and less able to resist cold,the rickety child is specially liable to this disorder.In consequence of the reduced capacity of the deformed thorax,even a slight catarrh,such as would give little trouble to a healthy child,may cause severe dyspnoea and cyanosis.Further, the weak state of the respiratory muscles readily allows of the occurrence of collapse of certain parts of the

tissue of the lungs by blocking of the bronchial tubes connected with them.The physiological and mechanical effects already described also manifest themselves in the production of anomalies of the lymphatic glands. The swellings of these found in rickets depends on the morbid changes of the intrathoracic organs;that of the lymph bodies of the neck on the irritation caused by the nasal and pharyngeal catarrh,and by eczema of the head and face;and so on with regard to implication of other local lymphatic structures elsewhere. Tuberculosis is also not infrequently associated with rickets,especially is the former found in connection with the latter in childhood.The,at first active,later on passive,hyperaemia of the rachitical bones,mainly of the peiphyses,predisposes to the local invasion of the tubercle bacillus as it floats in the circulation. In the lungs the disposition of the bacilli is as marked.Broncho-pneumonia is likewise a common complication of the rachitical condition.

CIRCULATORY SYSTEM.

When there is great deformity of the chest,complications of the circulatory organs are of common occurrence,and of a mechanical nature.The heart,crowded to one side,compressed and impeded in its movement, contracts rapidly and irregularly,with the result that one observes palpitation,tachycardia,and arhythmia; sometimes there is hypertrophy of the organ.In rare instances one finds at the autopsy milk spots on its pericardial covering,the evidences of compression exercised by the deformed bones.The arteries are usually of larger size than normal;and this leads to a lowering of the blood-pressure - particularly in distant parts,and also where normally the circulation becomes slow.This local stagnation of the circulation may have something to do with the hyperaemia in the rachiitical bones;and it certainly contributes also to the swelling of the viscera,predisposed as they are by the succulence and expansibility of their tissue. The feebleness of the general circulation and the swelling of the viscera themselves have also an effect, so that the condition in question is due to more causes than one .

THE BLOOD.

Rickety children not rarely suffer from anaemia, of the simple variety,when they have been a long time ailing and existing under unfavourable hygienic conditions.Auscultation will reveal in such children a systolic murmur at the base,with propagation into the vessels of the neck.It is also in these anaemic cases of rickets that we sometimes hear a systolic murmur, analogous to the carotid souffle,in auscultating the anterior fontanelle:this has already been referred to. These children are pale,weak,flabby,breathless on the slightest exertion;some are thin,others in good flesh; all have deformed and softened bones incapable of supporting the weight of the body.The anaemia of rick- etc may be complicated by hypertrophy of the spleen. Acute splenic anaemia has been fully described by Carr, Calcott Fox,Luxet,and others.The red corpuscles are less numerous than in health,the white are more

abundant,and the blood contains nucleated red corpuscles.So far as numbers are concerned,it may be noted that Luzet found a diminution of erythrocytes,within three weeks,from 2,110,000 to 1,596,000;Von Jaksch, within three months,from 1,600,000 to 750,000 red cells in a cubic millimetre of blood.In all of these cases the percentage of haemoglobin seems to be diminished to 30 or 60 per cent.,and the number of leucocytes increased to 25,000 or 36,000.This increase is caused by polynuclear and neutrophile cells,and does not depend on complications,for instance,with intestinal disorders.Hayem found only 658,000 red cells in an infant of two months;still,recovery took place.Cabot (A Guide to the Clinical Examination of the Blood,1897,p.105) in one case of rickets met with myelocytes,which are mostly found in the blood of malignant disease,but do not justify,through their mere presence,an absolutely bad prognosis.Lymphocytosis is prevalent,but,as Cabot justly remarks (Op. cit.,p.280),it is to a certain extent the normal condition of an infant's blood,provided that mononuclear - young - leucocytes are not in such excess as in the case of Rieder's,where 75 out of 100 leucocytes were of that nature.J.L.Morse (Study of the Blodd in Rickets,Boston City Hospital Medical and Surgical Reports,1897) concludes that the majority of rachitical cases "are accompanied by anaemia.This anaemia may be,or may not be,accompanied by leucocytosis.Leucocytosis occurs more frequently in the cases with splenic tumour than those without.It may be due to an increase of any or all of the varieties of white corpuscles.The specific gravity varies with the amount of haemoglobin.Finally,there is no form of anaemia found in rachitis which may not be found in other conditions,and no form of anaemia found in other conditons which may not be found in rachitis". The truth of this statement is apparent from the fact that every general constitutional disorder is bound to interfere with the proper manufacture of the blood,and in rickets the part so concerned are certainly implicated in the dyscrasia.

MUSCULAR SYSTEM.

In the subjects of rickets the muscles are pale, and,now and then,fat is found deeply deposited between the fibres,the microscopical structure of which appears to be normal.Nor is there a chemical change,though olein was missed by Jenner.The frequency of traumatic joint inflammain in infance and early childhood results in part from the incompetency of muscular resistance.Falls are very frequent,for the joints are flabby, the muscles being elongated as in poliomyelitis.The assertion of Kassowitz,that the ligamentous insertions are always inflamed,is not generally taken as proven. In the infant and in the newly-born the muscles are feeble.Their total weight,compared with that of the adult,is 1 to 40,while the relation of the skeleton is 1 to 26.It takes some time before the dynamometric effort of the child's muscles will increase.This takes place after the sixth year,but then it is only temporary and not persistent,nor is the sustaining and persistent strength of the muscle satisfactory,

perhaps in consequence of the relative absence, in the rachitical bone, of phosphoric acid and calcium. Experience certainly does not prove that weakness and atrophy of the muscles result from uneasiness or pain in any number of cases. Strabismus is common in small children simply because their muscles of accomodation and motion are insufficient, and more in rachitical children than in normal ones. Scoliosis is frequent in infants and children not suffering from, or affected by, any ailment except rickets. The so-called "growing pains" are often muscular only, and the result of over-exertion. What has been called rachitical pseudo-paralysis, by Berg and others, is but a confirmation of the fact that the muscular structure is insufficient. This condition may be universal, or only a certain number, or combination, of muscles are the principal sufferers. In some of these exclusively muscular cases ~~there is~~, as instanced by the case of Caillé's, there is a motion of the head in the same, or the opposite, direction. With strabismus, or sometimes without it, nystagmus, being in such cases the result of insufficient accomodation, is mostly noticed to be bilateral. There appears to be no gainsaying the fact that rickety children are apt to be strong after recovery, but the pressure they are subjected to in school, the expensiveness of fresh air in large cities, and the exposure to indoor life, one-half or more of the year in our climate, allow, when the spring does come, but little relief to the sufferers from rickets. Habitual scoliosis of the very young, up to the tenth year, is almost exclusively muscular, mostly dorsal, sometimes lumbar, with the convexity usually to the left. Rachitical babies, particularly when carried on one arm only, are very liable to grow scoliotic. Even when sitting in their chairs, not supported by their own strength, they topple over in one direction or another. Even before scoliosis occurs, there is a general flabbiness of the muscles which prevents free sitting altogether, and causes an apparent, or an actual, kyphosis. The latter, however, is easily distinguished from spondylitic kyphosis, inasmuch as it does not exhibit the same angular shape. As long as no bones participate in the deformity, the diagnosis can be easily made between that caused by weakness and that resulting from actual bone disease. The apparent curvature depending on the weakened muscles will instantly disappear, and lordosis, rather than kyphosis, will be observed on raising the infant's heels and hips while the chest and chin are supported. When intensified by the malnutrition of rickets, the feebleness of the infant's muscles is evidenced best of all by the symptoms connected with the insufficiency of intestinal muscular tissue in early life, which may be exemplified in a variety of ways. For example, one of the reasons why renal disorders are not at all uncommon in the intestinal diseases of early life - the others being the disproportion of the large renal arteries and the small capillaries, and the large size of the intestinal vessels and villi - is the feebleness of the intestinal muscle in the young, which is les capable of expelling decomposing faeces and toxines. This feebleness of the muscles shows itself, however, still more frequently in another symptom,

which is quite common in rachitical children,and even a diagnostic sign of early rickets.This is constipation.It is necessary to be able to exclude every other cause of constipation in order to hope to arrive at a diagnosis of rickets from this symptom;for instance, chronic inflammation of the colon and peritonitis, deficient or viscid mucus,local atrophy of the intestinal muscle,or stricture of the intestine,perhaps even the rare possibility of cystic neoplasms.The apparent constipation which results from insufficient feeding,either intentional or not,resulting in starvation;the superabundance of casein in the milk,of starch in artificial food;the relative absence of sugar; hardened faeces in the colon;hydrocephalus,and other causes of defective innervation;the drying up of the intestinal contents by excessive perspiration during hot summer months,in hot rooms,under heavy clothing,or by diabetes insipidus,- we shall,by the effect of antirachitical treatment,be able to arrive at a diagnosis of rickets,especially if time to taken to watch the other symptoms of that condition from time to time appearing.The debility of the muscles of the abdominal walls also,however,contributes to constipation;and the two together give rise to the flabbiness and inflation of the abdomen of rachitical infants. Some deformity of the abdomen,however,may be due to the spleen,liver,or kidneys.When Harrison's groove (a horizontal depression laterally above the diaphragm)is marked the liver and spleen are liable to be displaced, so that these organs may appear larger than they really are.Displacement of the kidney may also occur;and it is in children who have been or are rachitical that the majority of cases of floating kidney are seen at an early age.

DIGESTIVE SYSTEM.

Digestive disturbances are common in the rachitical subject.The tonsils are often large.The tongue is not usually coated to an unusual degree.On it are found little islands,red,marginated,deprived of epithelium. They will increase in size and number,and extend backward.They will heal and reappear.They are by no means syphilitic,as Parrot would have it,and correspond exactly with the erosins near the solitary glands and those of Lieberkühn in the intestine,which mean nothing worse than incompetency of absorption in that locality, and abnormal secretion.The stomach is in a condition of chronic catarrh,sometimes dilated.Acid dyspepsia is frequent.Anorexia and bulimia will alternate.The faeces contain an abnormally large amount of lime.Diarrhoea and constipation will follow each other at short intervals.The former owes its origin to faulty ingesta or chronic catarrh;the latter,sometimes to improper food, but more generally,as we have already seen,to muscular insufficiency.That the liver is usually,or at any rate, frequently enlarged and fatty,has been affirmed by many writers of repute.Observations on the living subject are not of much value as evidence n this point;there are many difficulties in the way of a thorough examination of the liver,and erroneous conclusions as to its true size may be easily arrived at.The meteorism which is so commonly present is enough to make any accurate

determination of the hepatic outlines,by percussion and palpation,impossible;for the liver is pushed upwards and backwards,besides being twisted on its horizontal axis.In this way,we may be led to believe that the liver is smaller than it really is.On the other hand,serious narrowing of the chest may cause downward displacement of the organ,and a delusive semblance of enlargement.But,even if we set aside the difficulties in the way of physical examination,we have to bear in mind that in young children,especially when at the breast,a certain degree of fatty liver is normal;and that the organ may thus exhibit - apart from any disease - a volume relatively greater than in the adult.Lastly,the disturbances of the respiratory function that are so common in rickety children,and the venous stagnation to which they give rise,may,from passive congestion,cause enlargement of the liver. Accordingly,we are justified in concluding that clinical observation furnishes no certain proof that the liver is abnormally infiltrated with fatty matter in rickets.Post-mortem inspection,again,discloses a considerable degree of hepatic enlargement in a small minority of cases only;and here it may be noted that the death of rickety children often occurs under circumstances of extreme emaciation,due to disordered nutrition;and that the fatty liver,in such cases, possesses the same significance as in all other forms of wasting disease,and stands in no special relation, even when it exists,to rickets.When rickets is superadded to some constitutional disorder of a chronic character (e.g.,when it comes on after recovery from inherited syphilis),or when it is due to hereditary predisposition,enlargement of the spleen appears to be one of its earliest symptoms;and,as in other diseases associated with a dyscrasia,is usually due to corpuscular overgrowth.Extensive follicular ulceration of the intestines,tuberculosis,etc.,may lead to the comparatively rare condition of amyloid degeneration of the spleen.In a large proportion of rachitical cases the splenic enlargement may be demonstrated by percussion,and,still more surely,by palpation.The rounded anterior edge of the organ may often be felt under the costal margin,or even well in front of it,over a considerable area;so that,in spite of the meteorism,we may conclude that the spleen is positively enlarged. Ritter v.Rittershain found a marked degree of splenic enlargement in ten out of thirty-five post-mortem examinations,and the frequency of its occurrence has been dwelt upon by other writers since his time.

INFECTIOUS DISEASES.

The possibility of tuberculosis as a complication has been already alluded to:in fact,this accident very often carries off rickety children.It is met with in them in any of its usual localizations,such as the lungs,bones,articulations,meninges,skin,etc.Hereditary syphilis is another aggravating cause which weighs heavily upon the subjects of rickets.Scrofula is less serious,and we often see rachitics recover perfectly from otorrhoea,blepharo-conjunctivitis,keratitis,etc. In the event of an infectious disease,such as measles, diphtheria,scarlatina,pertussis,typhoid fever,influenza,

or tuberculosis attacking a rickety patient,it is of much more gravity than when it occurs in a child who is not so afflicted.By reason of the violence of its spasmodic seizures,whooping-cough threatens the life of the rachitic child with the deformed thorax,disposing to asystole.Of itself rickets is a curable affection;but,when complicated with a general infectious disease,it is often fatal,the termination of such diseases depending,in very great measure,upon the soil (that of the rachitic being very favourable) upon which their respective p athogenic micro-organisms become engrafted.

A C U T E R I C K E T S.

The condition which is so designated has often been described,and also as multiple epiphysitis,or multiple periostitis of the articular ends of the long bones.The changes which,in the usual form of rickets require months to develope,take place in a very short time.Not infrequently the children were quite well before they were taken with this peculiar affection. Cases have been known to occur between the fourth and and twenty-fourth months of life,and to last from two to six weeks,or just as many months.They have been known to get well,or a few of them terminate fatally. They are accompanied with fever and rapid pulse,perspiration,and,now and then,diarrhoea,with eager or reduced appetites.At the same time the epiphyses swell very rapidly,and are painful.The same is true of the diaphyses and the flat bones of the head.Many authors do not recognize this form as an independent variety.Some call it an acute initial stage of certain cases of rickets,as they are not infrequently found in infants which exhibit a very rapid growth.Some have taken it as an independent disease,developed on the basis of a constitutional disposition;some look upon it as a very intense acute form of rickets;others,as an inflammation of the bone.Some refer it to hereditary syphilis,and a few to the influence of malaria. That the disease is epiphysitis and periostitis there is no doubt.Th.Barlow has reported cases complicated with subperiosteal haemorrhages.I Do not hesitate to claim the condition in question as rickets,for epiphysitis and periostitis of early age,not of rachitical basis,are not apt to run such a favourable course as this form frequently does.Had all the cases of other observers been seen and studied,the differences of opinion would probably not have been so great.It will not do to judge of unobserved cases by the light shed by a single case under one's own observation.One may see cases of acute rickets which were in the initial stages of general rickets,and have observed those of local or multiple epiphysitis,mainly after infectious fevers,which were diagnosed as such.They are,however, very uncommon.But,even with preceding infectious fever, such as scarlatina or,more frequently,typhoid fever, there are unexplained cases of rickets and deformity. Thus,R.Barwell (Trans.Path.Soc.,xxxiv.,pp.203-208) exhibited some before the Pathological Society of London,which are positive proofs that some forms of ostitis may occur,and result in the most formidable deformities,without being rachitical.A girl,of seventeen years,was perfectly well formed up to the age of two and one-half or three years.After that time the deformities began to develope,and did not change after she was thirteen;at which time her left humerus

measured 7¾ inches from the shoulder to the elbow (distance 6¼);the right humerus 7½ inches (distance 4¼ inches);the left tibia 10 inches from knee to ankle (distance 7¼ inches);and the right tibia 9½ inches (distance 4½ inches).The patient's bones were always very brittle.When she was between nine anf thirteen, she broke her arm four times,and her lower limbs on several occasions.Another patient was a male,of twenty-two years,who was born healthy and well formed, continued thus until five years of age,when he was attacked with a fever,after which the bones became soft and bent.Osteotomy was performed on him,and the femora were found to be mere thin shells of bone,surrounding cavities containing great quantities of medulla, which flowed out of the wound like oil;five ounces were discharged at once.In both cases there appeared to be an hypertrophy of the medulla at the expense of the bone-substance - a condition which Barwell proposes to call eccentric atrophy."While these subjects are still youthful very little bone-earth is deposited,or at least remains in the very thin layer of osseous tissue that subsists.The relationship between infantile ostitis and extreme development of the intraosseous fat,though well known,is still occult;neither should we lose sight of the possibility that the softening process of ostitis may be due to a fatty acid.Now,fatty ostitis usually occurs in epiphyses." According to this author,the shafts participated in the affection.

FOETAL RICKETS.

This variety of rickets has also been termed "foetal cretinism"and "achondroplasy".It was said to be very rare by Virchow and Ritter;on the other hand,frequent by Kassowitz and by Unruh.In 1887, Schwartz claimed that 400 out of 500 babies born in Briesky's clinic were rachitical.The cause of these discrepancies must be sought for in the fact that writers are not unanimous in their opinions as to their cases.They are not alike.The majority of those one sees are decidely cases of rickets,in which formerly normal bone had become diseased.A small number of others have been described in which the osseous system was thoroughly decalcified,where torsions of limbs and fractures of bones were found in a large number of places,which undoubtedly were the result of imperfect osteogenesis,such as Stilling described in 1889. E.Kaufmann (Investigations on the So-called Foetal Rickets,Berlin,1892) removes faotal rickets altogether from what is called rickets in after life.The cases thus far reported have a common basis - i.e.,defective ossification.He distinguishes three varieties - the malacic,hypoplastic,and the hyperplastic.In the first, cartilage remains have become softlin the second, growth is interrupted,arrested;in the third,grwoth is lively,but inordinate.The final outcome of the process depends on the period of its starting.When this takes place late,the foetus may live with more or less deformity of head,trunk,limbs,ar more or less damage to intelligence.O.Lubarsch (Results of General Etiology in the Diseases of Man and Animals,Wiesbaden,1896) collected 21 cases.Some of them were idiotic,some not at all.The extremities were straight,or curved, or

shortened;the sexual organs were infantile,or fully developed.Some cases were hereditary.The skeleton was often infantile,the bone-forming cartilage persisted, and was sometimes present in small quantities only. Normal ossification,that would take place in this small amount of cartilage at any one time -e.g.,at the base of the skull,would reduce the length of the latter. Ossification,taking place prematurely in fully developed bone-forming cartilage in the same place,would have a similar effect.If,during foetal life,it would cripple the base of the brain by shortening the base of the skull.In this way different forms of cretinistic,cretinoid,and cretin conditions (Virchow) would depend - either on the amount of the bone-forming cartilage,or the period of its change into bone,or on both.The same effect would result from the same changes when taking place in the foetal vertebral column,and in the extremities,or in the pelvis.Those extremely rare cases of foetal rickets,which are described as something absolutely different from all other forms,are probably instances of the malacic form of the disease.The hyperplastic malnutrition of the cartilage - chondro-dystrophia - is represented in dwarfism,with its persistence of all cartilage,and when the thyroid gland is inactive,or absent at the same time,in the so-called congenital myxoedema.The hyperplastic chondro-dystrophia - resulting in premature ossification of the basis cranii and shortening of the base of the brain,with retraction of the vomer,and root of the nose,and with flat palate,with or without the co-operative influence of an endemic maism,and with or without the absence of thyroid function - terminates in cretinism complicated with more or less dwarfism. From this point of view the cases of Siegfried Müller (Periosteal Aplasia with Osteopathyrosis under the Guise of so-called Foetal Rickets,Munich,1893),and John Thompson lose their mysterious aspect,and gain in interest.Müller describes a case in which the proliferation of cartilage rendered the periosteum atrophic,thereby crippling the after-growth of the bone;and another case in which,vice versâ,the cartilage was rendered atrophic by the proliferating periosteum - foetal chondro-malacia. The patient was a twin whose brother was healthy;and the case of Klein's, which he cites,was of the same nature.Kaufmann tells of a case which resulted from incest between sister and brother;and Porak one which occurred in a mother and child.The cases reported by John Thompson (Note on Three Living Cases of Achondroplasia:Chondro-dystrophia Foetalis,or so-called Foetal Rachitis,Edin.Med.Jour., June,1893) were those of a boy of five,and of two men, of thirty-nine and thirty-six years,all the other members of whose families were well.What is classified above he defines as "absence,arrest,or perversion of the normal".In his cases "those bones which are formed entirely in membrane,and those which,though formed in cartilages,remain altogether or mainly cartilaginous till a late period of intra-uterine life,and found quite normal in size" (upper part of cranium,neck, trunk,clavicle,scapula,sternum,spine,carpal,and tarsal bones);"those depending on endochondrial ossification for their growth in utero" (the long bones of the

extremities,base and skull,ribs,and pelvis)"are dwarfed.

L A T E R I C K E T S.

Cases of retarded rickets (rachitis tarda) are those exceptional ones in which the disease persists very long and appears at a late date.Recovery,in the usual run of cases, may be late,and ossification very much delayed.The large fontanelle may remain open to the tenth year,or even longer;the temporary teeth may remain beyond the usual period,and when they fall out they are not replaced in due time.Some such children cannot walk,because of the softness of their bones, until they are eight or ten years old;or,in otherwise healthy children,rickets will break out in advanced age,bones will soften,knock-knee make its appearance. Some such cases have been claimed as osteomalacia,if, indeed,it is possible to differentiate this from rickets,except through the fact that it appears in the fully developed;rickets,however,in the growing bone.At no age should we expect all of the possible symptoms of rickets to occur together and uniformly. Rehn noticed at an advanced age softening of the diaphyses,with but littleepiphyseal swelling.Bierdert the same,together with softening of the ribs and the epiphyses.Local softening has been principally observed in the neck of the femur:in such cases,however,we ought not to forget that in many instances of early rickets the femoral neck is often detached by rickets, and that it takes a long time to complete the ossification of the head and neck down to the trochanter. J.Schulz describes the case of a girl of fifteen,who complained since her third year,and had a prominence and elevation of the trochanter.Deformities of the same neighbourhood were noticed by E,Müller - in adolescents of sixteen,seventeen,eighteen,and nineteen years.Bilateral rachitical curvature of the neck of the femur was noticed in a boy of fifteen,with reclining gait,by Rotter.Royal Whitman had four cases,in boys of fifteen,sixteen,eleven years,of bending of the neck of the femur.He accuses rapid growth,overwork, standing up,and carrying loads as predisposing causes. B.F.Curtis published a similar case,in a boy of seventeen,who,without other marks of rickets,had complained,for fifteen months,of pain in the right knee,and two months of walking lame.There was no sign of tubercular mischief in the bone at the operation,only a bending due to rickets.The usual shortening remained after recovery.

D I A G N S I S.

From the description given above it should be easy to recognise rickets.Indeed,when it presents its usual epiphyseal swellings,and other well-known phenomena,the diagnosis is obvious.Still,the partial forms,limited to one member or bone,the intiial stages,and the attenuated forms often require careful consideration before an accurate diagnosis can be established.It may happen that a child,previously in good health,developes weakness,malaise,and pallor; it is less lively than usual,sinks down in the arms of its nurse,and refuses to stand;it is tired,inert, without elasticity,and bereft of energy.If with these symptoms we note that its alimentation leaves much to be desired,that the eruption of its teeth is delayed, and that the anterior fontanelle does not close,we have to fear the onset of rickets.In every poorly nourished child,either bottle-fed or prematurely weaned,rickets is imminent,and we ought to be on the watch to seize the first opportunitymof dealing with the premonitory signs of the disease.These signs,it is true, may be fleeting,and the disease may become arrested in its progress,recede,and disappear;but an attentive and intelligent observer will not be deceived.We must then be able to recognise the slight forms of rickets in order to attack them early,and so save the child from the severe troubles which would not fail to arise if the malady escaped recognition.The general invasion of the system must be fought against,as the disease progresses very slowly.Serious difficulties of diagnosis may arise if the disease is partial,and limited to one member or bone.Given a deformity of the vertebral column,a gibbosity of short radius,with weakness of the lower extremities,which are,however, straight,the rest of the skeleton appearing to be normal - the question arises as to the condition being one of kyphosis or of Pott's disease.If the kyphosis is frankly angular Pott's disease is evident,the projection of a vertebra backwards bearing witness to the destruction of the vertebral column segments.The rachitic gibbosity has a short radius,it is rounded and not angular.But there are cases in which doubt is admissible,and in which we must examine the spinal column with the greatest care,study its degree of mobility,palpate,and percuss the spinal processes and the lateral depressions,and investigate the sensibility and motility of the lower limbs.When it impossible to make up one's mind on the spot,it is advisable to continue the diagnostic search at a later date.Scoliosis (lateral deviation of the spinal column) can scarcely be mistaken for Pott's disease;the former,in the rachitical infant is apparent only,and depends on muscular debility,like kyphosis,which,when rachitical only,is,in the young, recognized by its disappearance when the infant is supported by its chest and heels.Another condition which may be mistaken for rickets is congenital

dislocation of the hip;the children affected with it walking with the knees turned in - knock-kneed.But it will suffice to search for the head of the femur in order to determine that it does not occupy its ordinary position,but is in the external iliac fossa above the cotyloid cavity,and thus establish the diagnosis. We must not forget,however,that rickets may coexist with congenital disclocation of the hip,as also with Pott's disease,and we must be able to distinguish the symptoms belonging to each.We think immediately,and usually with reason,of rickets when a child does not begin to walk until late,or walks badly.However, walking may be retarded or prevented by other causes, by atrophic infantile paralysis,which may be recognized by its characteristic course,by its localization in certain groups of muscles,and by the accompanying atrophy of these muscles.Obstetrical paralysis and syphilitic pseudo-paralysis may also delay walking. Rickets must be distinguished from hereditary syphilis of the bones when it happens to be limited to the lower extremities.The rachitical curvatures of the long bones are quite characteristic.If they be combined with swellings,the latter belong to the epiphyses,and are not to be compared with the gummatous periosteal irregularities sometimes noticed in syphilis.The latter are surrounded with oedematous soft parts,are steep,circular,and even spindle-shaped. Sometimes,however,we find rickets and syphilis existing together.In both the localities of active growth are affected;in both the transportation of calcifying cartilage into bone is arrested;and in both there is ample proliferation of cartilage cells.The peri-epiphyseal lines of the extremities suffer in both. Disruption between epiphysis and diaphysis - more in the upper end of the humerus than in other localities - and suppuration of the joints may take place in syphilis,but not in rickets.Craniotabes is characteristic for rickets;in hereditary syphilis the cranium is rarely affected,and mostly at a later period,and the changes are granulomata,mostly on the parietal bones.The syphilitic tibia,called also the Lannelongue tibia,is a bone which appears to be curved,but which is simply swollen,nodulated,of uneven surface, deformed by gummatous deposits,osteophytic layers, etc.,The rachitic tibia,on the contrary,is convex anteriorly,concave posteriorly,flattened laterally, and actually curved;it has the shape of a cavalry sword.In addition to thes objective symptoms,which are sufficient for a diagnosis,we have,betraying the presence of hereditary syphilis,Hutchinson's sign in the teeth,the cutaneous syphilides,and other phenomena in connection with the eyes,ears,and other parts.In the event of rickets being limited to the head,one may fall into the mistake of diagnosing it as hydrocephalus - a disease characterised by an extraordinary development of the caranium,by a widening of the sutures and of the fontanelles,by idiocy,and so forth.In hydrocephalus the cephalic souffle is rarely perceived on auscultation of the anterior fontanelle,the extremities are not deformed,and the face contrasts,by its smallness,with the enormous

enlargement of the cranium.Rickets may,however,be complicated with a certain degree of hydrocephalus, which may be recognised by the occasional presence of craniotabes,the widening of the fontanelles,and by the large size of the head.Finally,it is a more difficult matter,though one of great importance as regards treatment,to recognise the first beginnings of the disease.The disorders of digestion which usually precede the changes in the bones,especially in young children,are not in themselves characteristic of rickets;but when they occur during the second half-year of life,about the time of the first dentition, and show an obstinate tendency to recur,they ought to make us suspect the approach of rickets,whose earliest specific signs will be furnished by the delay and irregularity in teething,the local sweating of the head,and the behaviour of the sutures and fontanelles - all of which phenomena have already been dwelt upon. To quote Holt: "The most important early symptoms for diagnosis are swesting of the head,craniotabes,great restlessness at night,delayed dentition,and enlarged fontanelle.All these,taken separately,may means something else,but collectively they can mean nothing more than rickets.

P R O G N O S I S.

The possible outcome of rickets will depend upon the course and severity of the disease. It always lasts - apart from those rare cases of acute rickets, which run their course in a few weeks, and which have already been alluded to above - for months, or even years. The earlier the age at which it sets in, the more rapid and severe are the changes wrought in the bones, and the associated troubles, - especially those of the respiratory organs, - and the more profound, accordingly, the interference with the child's general health and nutrition. That form of rickets which begins during the second year, or even later, runs a more sluggish course; it is not attended by fever, or by any considerable degree of constitutional disturbance. Its course is often interrupted by pauses of variable length, during which the bones cease to swell, the digestive troubles subside, and the nutrition improves in consequence; restlessness and fever, and also exacerbation of the gastric disorders, generally heralding the resumption of morbid action. In almost every instance those cases of rickets that begin late and run a slow course end favourably. There remain a certain plumpness of the affected bones, more or less deformity, and permanent stunting of the body. When the disease sets in early - i.e., during the first twelve months, it may also terminate in recovery; this usually sets in with improvement in the child's nutrition and general state; the teeth come through more quickly, the swollen epiphyses subside, and the bones become consolidated. But many of the younger sufferers never live to reach this stage; they are destroyed by one or other of the complications enumerated above, especially by disorders of the respiratory system. Since the latter are most prevalent during the cold season, this is the most dangerous time of the year for rickety subjects. In summer, again, they are liable to gastro-intestinal catarrh, and this is a frequent cause of fatality. Mild forms of rickets are cured rather promptly without complications and without difficulty, provided that the significance of their symptoms be understood, and that their cause be recognised, and, consequently, be removed by change in diet and in general hygiene. The moderately severe forms are also curable, but less rapidly; a period of three, four, or six months is necessary to effect a cure of moderately severe rickets. When the disease is partial, when the deformity is restricted to one member (genu valgum), to the head (craniotabes), or to the trunk (scoliosis or kyphosis), the termination is hardly more rapid than in the generalized forms. The intensity and the depth of the lesions are more important than their extent, or their dissemination. The more the process of ossification is disturbed the slower the cure. In many cases, even of partial rickets, surgical intervention is necessary, owing to the futility of internal medication. Treatment is also often next thing

to ineffective in the severe cases of rickets with extensive deformities, the surgeon's services being here likewise required. In the case of women the prognosis is more grave because of the deformity of the pelvis - a much-feared cause of dystocia, which may necessitate symphyseotomy, cephalotripsy, Caesarean section, or other obstetrical operations, more or less dangerous for mother and child. Inasmuch as rickets does not directly compromise existence, it is not a fatal disease. Relatively good, therefore, as regards life, the prognosis is bad as regards the duration of certain visceral manifestations of the dyspepsia, which, having preceded the bony lesions, survives them. In mild cases the gastric phenomena are slight, the rachitical dyspepsia being themore marked and persistent in proportion as the rickets itself is more pronounced.

T R E A T M E N T.

In dealing with a case of rickets one's chief duty is to lay down certain preventive measures or hygienic rules, so that the malady may be either obviated, relieved, or shortened. Now, if the original disposition to rickets, as has been suggested, is to be looked for in early intra-uterine life, when the blood-vessels begin to form and to develope, we know of no treatment directed to the pregnant woman, or uterus, which promises any favourable result. But, the more we recognize an anatomical cause of the chronic disorder, the more we can appreciate the influence upon the child of previous rickets in the mother, and we are justified in emphasizing the necessity on the part of the woman to be healthy when she gets married, and to remain so while she is pregnant. After the child is born the most frequent cause of rickets is found within the diet or the digestion of the patient. To attend to the former is, in almost every instance, equal to preventing disorders of the latter; for most of the digestive disturbances during infancy and childhood are the direct consequences of errors in diet.

The main thing, then to be attended to is the feeding of the infant. We know that improper feeding stands foremost among the causes of those gastro-intestinal disorders which are so dangerous to infant life; and, in speaking of the etiology of rickets, I pointed out the intimate connection between these disorders and the malady in question. During the first few months after birth, these disorders may be prevented by keeping the infant at the breast; should the mother be unable to suckle her own child, a good wet-nurse should be provided. The milk of the mother should not be dispensed with when there is the slightest opportunity to obtain it, particularly when the family history is not good, and nutritive disorders are known to exist, or to have existed, in any of its members. When it cannot be had, artificial food must take its place, and it is in the selection of it that most mistakes are constantly made.

This much is certain,that without animal's milk no infant can,or ought to,be brought up;as ass's milk can be had only exceptionally,and dog's milk,which has been said to cure rickets,is still less available, the milk of either goat or cow must be utilized.The former ought not to be selected if the latter is within reach,mainly for the reason that it contains, besides other objectionable features which it possesses in common with cow's milk,an enormous percentage of fat.Cow's milk differs in this from woman's milk,that it contains more fat,more casein,more potassium,and less sugar than the latter,and that its very casein is not only different in quantity,but also in chemical properties.Even the reaction of the two milks is not the same,woman's milk being always alkaline being always alkaline,cow's milk often either neutral or amphoteric,and liable to acidulate within a short time.Thus,the dilution of cow's milk with water alone yields no equivalent at all of mother's milkmthough the dilution be large enough to reduce the amount of casein in the mixture to the requisite percentage of one,and one only,in a hundred.The addition of loaf-sugar,and of table salt,and sometimes bicarbonate of sodium,or such an alkali as lime-water,according to special circumstances,is the least that can be insisted upon.Besides,the cow's milk must be boiled to prevent its turning sour too rapidly,and this process may be repeated with advantage several times in the course of the day.Instead of water,some glutinous substance must be used for the purpose of diluting the cow's milk.As its casein coafulates in hard,bulky curds,while woman's milk coagulates in small and soft flakes,some substance ought to be selected which keeps its casein in suspension and prevents it from cudrling in firm and large masses.Such substances are gum-arabic,gelatine,and the farinacea.Of the latter,all such must be avoided which contain a large percentage of starch.The younger the baby,the less is it in a fit condition to digest starch;thus,arrowroot,rice,and potatoes ought to be avoided.The very best of all farinacea to be used in diluting cow's milk are barley and oatmeal.A thin decoction of either contains a great deal of both nutritious and glutinous elements, the former to be employed under ordinary circumstances, the latter to take its place where there is,on the part of the infant,an unusual tendency to constipation.The decoction may be made from one to three teaspoonfuls of either in a pint of water,boiled with a little salt,stirred,from ten to twelve minutes,and strained through a coarse cloth.It ought to be thin and transparent.Then mix with cow's milk,in different proportions according to the age of the baby.Four parts of the decoction,quite thin,and one of milk (always with loaf-sugar),for a newly-born,equal parts for an infant of six months,and gradual changes between these two periods will be found satisfactory. Whenever there is a prevalence of curd in the stools, the percentage in the food of cow's milk must be reduced,and,now and then,such medicinal correctives resorted to as will improve a disturbed digestion. Care ought to be taken lest,for the newly-born or quite young,the preparations of barley offered for sale contain too much starch.The whiter they are,the

more unfit for the use of the very young,for the centre of the grain contains the white and soft starch in preference to the nitrogenous substances which are found near the husk.Thus,it is safest to grind,in one's own domestic coffe-mill,the whole barley,but little derived of its husk,and thus secure the most nutritious part of the grain,which is thrown out by the manufacturer of the ornamental and pretty packages offered for sale in our shops.But few cases will ever occur in which the mixture above described will not be tolerated.In a few of them,in very young infants,the composition recommended by Meigs (Med. News,Nov.28,1882) proves successful.It consists of three parts of a solution of milk-sugar (3xvii¾ in Oi of water),two parts of cream,two of lime-water, and one part of milk.For each feeding he recommends three tablespoonfuls of the sugar solution,two of lime-water,two of cream,and one of milk;mix and warm.One-half,three-fourths,or all of it may be taken by the infant.The subject might be pursued further,and a good deal be said regarding milk laboratories and percentage feeding,now so popular in certain large cities where such institutions have been established, but it is obviously out of place to write an essay upon infant feeding in connection with the malady under consideration.Suffice be it to indicated broad general principles as hereon;which same are based on a not inconsiderable experience,and specially recommended for the simplicity and facility of preparation of the articles.The substitutes offered for sale,under the title of infant foods are in part worthless,all of them expensive,when compared with the simple articles recommended above,and not recognizable as to their uniformity and compounds.But,no matter how appropriate a mixture,such as the above described,may be,it is always for the young infant to be considered as a makeshift.It is to be used as a representative of mother's milk only when this cannot be had.Therefore, it is better to alternate with breast-milk when this is secreted in but an insufficient quantity.Some good breast-milk is better than none at all;but with this proviso,that it is g o o d.There are some milks either too watery,or too dense and white.The former will produce diarrhoea,the latter hard and dense curd.The former may be improved by feeding and strengthening an anaemic and overworked mother;the latter,by giving the baby,before each nursing,a tablespoonful of a mixture of barley-water and lime-water,or,when it produces constipation,lime-water and thoroughly sweetened oatmeal-water.The cases in which breast-milk,such as can be had,is not digested by the infant are rare,but they will occur.In them the proper substitute will yield a better result than mother's milk;for the mother's milk will not always be a boon,and must then be dispensed with.Particularly is this so when it is too old.Weaning ought to take place when the first group,or the first two groups,of teeth have made their appearance.After that time mother's milk is no longer the proper food,and,instead of preventing indigestion and sickness,it is a frequent cause of them,and of rickets.Instead of muscle,it will then give fat,and the large fontanelles and big head,the paleness of the rotund cheeks,the flabbiness of the soft abdomen and

thighs,will tell the tale of rachitical disease - slowly engendered by the persistent employment of an persistent employment of an improper article of food.

In early infancy digestive troubles are sometimes the result of primary gastric disturbances,which,of course,will present their special indications for treatment.A frequent occurrence,together with a general gastric catarrh,is the presence of fatty acids in the stomach,such as an improper amount of lactic, acetic,butyric,etc.,acids.Before digestion can be anything like normal,they must be neutralized.For that purpose calcined magnesia,carbonate and bicarbonate of sodium,prepared chalk,and lime-water have been found useful.The latter,as it contains but a trifle of lime, in order to neutralize,must be given in larger doses than is usually done;a tablespoonful contains but a quarter of a grain of lime.Furthermore all of the alkalies must be given,not in food only,but also between meals;for,when given in the former way alone, it neutralizes the abnormal and injurious acids,together with the normal digestive secretion,the lactic and muriatic.Not infrequently,when the infants have suffered for some time,general anaemia will set in, and result in diminishing the normal secretions of the mucous membranes and glands.In those cases which do not produce their own gastric juice in sufficient quantity or quality,pepsine and hydrochloric acid may be given to advantage.In those cases a very good plan is to add a fair amount of chloride of sodium (one-half to one drachm daily) to the infant's food. I.Rudisch (Amer.Jour.Obstet.,July,1879) mixes one part of dilute hydrochloric acid with two hundred and fifty of milk,and then boils (one-half teaspoonful of dilute muriatic acid,on pint of water,and one quart of milk). The addition of bismuth ot any of the proposed plans is admissible and beneficial,especially when there is erosion or soreness of the gastric mucous membrane. There are also cases in which wine and the bitter tinctures,which are known to increase the secretion of the gastric juice,render valuable service.Rachitical infants,at an early age,require to be given such substances,either dietetical or remedial,as are believed,or known,to add to the ingredients of the organism,in a form not requiring a great deal of change. Thus,meat-soups,mainly of beef,and of mutton,in complications with diarrhoea,ought to be given at once whenthe diagnosis of rickets becomes clear or probable.Any mode of preparation will prove beneficial; the very best,however,is to utilize the method used by Liebig in making what he calls beef-tea.A quarter of a pound of beef or more,tender and lean,cut up finely,is mixed with a cup or a tumbler of water,and from five to seven drops of bilute hydrochloric acid. Allow it to stand two hours and macerate,while stirring up occasionally.This beef-tea can be much improved upon by boiling it for a few minutes.It may be given by itself,or mixed with sweetened and salted barley-water,or the usual mixture of barley-water and milk which the infant has been taking before. Older infants,particularly those suffering from diarrhoea,take a teaspoonful of raw beef,cut very

fine,several times a day.It ought not to be forgotten, however,a tapeworm may be got by eating raw meat.The following if the dietary given by Louis Starr(Diseases of the Digestive Organs of Children) in cases of rickets uncomplicated with diarrhoea(age 18 months):

First Meal,7.30 a.m.- A breakfast cupful (8 ounces) of milk with a tablespoonful (one-half ounce) of cream; on alternate days the yolk of a soft-boiled egg,with a little butter,salt,and bread-crumbs,and two to four tablespoonfuls of well-cooked and strained cracked-wheat porridge with cream and salt.

Second Meal,2 p.m.- A good tablespoonful of well-minced andmpounded chicken or mutton,with gravy and a little crumbled stale bread;a tablespoonful of purée of spinach,stewed celery,or cauliflower tops;thin bread and butter.

Third Meal,6 p.m.- Milk and cream as before; thin bead and butter.

He avoids the use of farinaceous food,and allows pure water as a drink.He likewise includes in his diet-list a meal,at 11 a.m.,consisting of a breakfastcup-ful (8 ounces) of milk,with a tablespoonful (one-half ounce) of cream and a slice of whole-meal bread.In the event of the case being complicated with diarrhoea and offensive stools,he arranges the diet so as to contain a minimum amount of casein - thus:

First Meal,7 a.m.- Veal broth (half pound of veal to a pint of water) and barley-water equal parts (3 to 4 ounces).

Second Meal,10 a.m.- Cream,half ounce,whey (freshly prepared) six ounces.

Third Meal,1 p.m.- Same as the first,with chicken broth in place of veal broth.

Fourth Meal,5 p.m.-Same as the second.

Fifth Meal,10 p.m.,Same as the first.

He repeats the second meal at 4 o'clock in the afternoon in the event of the patient being feeble; and in extreme cases with diarrhoea restricts the diet to raw beef-juice in one to three tablespoonfuls per dose every two hours,with a modified brandy-and-egg mixture twice a day.For the manufacture of the latter Starr beats up well the yolk of a raw egg,adds ten drops of brandy,one teaspoonful of cinnamon-water, and one coffeespoonful of white sugar

In cases of rickets F. Mendel administers the extract of the thymus gland,and says that all the symptoms improve,the sweating diminishes,the bones grow stronger,dentition progresses,and the attacks of laryngismus stridulus become fewer.He employs the remedy in the form of tablets in soup,and has had experience of it in one hundred cases.Red bone-marrow has also been given in rachitical cases,in the form of the glycerine extract,one or two teaspoonfuls after meals,well diluted with water.

Some form of iron is usually indicated in rickets, save when our attention is claimed by some intercurr-ent disease,especially of the respiratory organs. The saccharated carbonate combines very well with the bismuth;a grain three times a day,or less,will answer well.The citrate of iron and quinine can be given for a long time in succession.When the case is complicated with glandular swelling,as it often is,the syrup of

the iodide of iron, in sweetened water or sherry, gives satisfactory results.

For the treatment of rickets Bretonneau and Trousseau were the first to recommend cod-liver oil; the yellow oil, containing the alkaloids, is to be be preferred to the white oils from which they have been removed. In general the amount should be rapidly increased until pretty large doses are being given. Carmichael, however, advised the exhibition of small doses, or even frictions with the oil, from which very little absorption could be expected. In order that the oil may be tolerated by very young children, it should be mixed with some syrup. The animal or vegetable fats, which several have advised as substitutes for the fish oil - such as butter, lard, tallow, olive oil, etc. - inasmuch as they contain no iodine, phosphates, or ptomaines, to which cod-liver oil owes so much of its beneficial action, are of no value whatever in rickets. Gautier and Mourgues explain the action of cod-liver oil as follows: 1. Cod-liver oil acts through its fatty bodies which are readily assimilable, thanks to their partial saponification by the action of the hepatic ferments, and by the dissolution of a certain quantity of biliary matters. The fatty bodies are the reserves which are stored in certain of the tissues, and are destined to be used by the economy to supply the need for calorification. They cannot, therefore, be replaced by less digestible fats or fatty acids. 2. This oil acts as a repairer of the tissue, in virtue of its richness in phosphates, phosphoglyceric acid, lecithin, and phosphorus in organic combination. The small amounts of iodine (3 or 4 cgm. per litre), or of bromine, also aid in this reparative action. 3. Finally, the oil acts by its alkaloids, which are found in the coloured oils and render them much more active than the white ones. These alkaloids of cod-liver oil have been isolated by J. Bouillot, and given in doses of 15 to 25 cgm. in the twenty-four hours. In the case of two children suffering from malnutrition, the appetite returned under this treatment in a few days. Cod-liver oil, the administration of which should be continued for a considerable p eriod, is indicated especially in cases of emaciated and cachectic children tainted with tuberculosis or scrofula. It is a trusted tonic in rickets, and will remain so. Animal oils are so much more homogeneous to the animal mucous membrane than vegetable oil that they have but little of the purgative effect observed when the latter are given. The former are readily absorbed, and thus permit the nitrogenous ingesta to remain in store for the formation of new tissue, but still affect the intestinal canal sufficient to counteract constipation. As the latter is an early symptom in a peculiarly dangerous form of rickets, craniotabes, cod-liver oil ought to be given in time. Diarrhoea is but seldom produced by it; if so, the addition of a grain or two of bismuth, or a few doses of phosphate of lime (one to four grains each) daily, amy suffice to render the movements more normal, There are very few cases that will not tolerate cod-liver oil at all. The pure cod-liver oil - no mixtures, no emulsions - ought to be given; the large quantities of lime added to it in the nostrums of the wholesale druggists embarrass digestion, and bring on distressing signs

of constipation.These mixtures have been prepared, and are eulogized on,the plea of furnishing to the bones the wanting phosphate of lime.The bones,however, refuse to accept the service so offered.

Phosphorus is a drug that has been much employed in the treatment of rickets,and apparently with good results,though Kissel states there is no evidence in favour of the use ofthe metal in this disease.Wegner (Virchow's Arch.,vol.50),when feeding young animals on minute doses of phosphorus,found the medullary spaces, which penetrate into the calcifying cartilage,to be fewer and smaller than in animals not so phosphorized. Whe he fractured the bones of rabbits,and fed the animals on phosphorus,the bones would heal in a much shorter time than the fractured bones of animals not so fed.Troussea was the first to try pure phosphorus in rickets.He prescribed it in the form of phosphorated butter (1 cgm.phosphorus to 300 gm.butter)spread on thin slices of bread.Swetchen and others have observed a cure result in many instances,and Kassowitz looks upon phosphorus as an absolute specific for rickets.It may be prescribed in the form of white phosphorus (gr.iss) and cod-liver oil (Oij),one to two teaspoonfuls in the day.Oil of sweet almonds may be substituted for the cod-liver oil.The mixture may be rendered palatable by syrups or aromatic substances, such as peppermint,aniseed,strawberry syrup,etc.The daily dose of phosphorus ought not to exceed 1/64 - 1/32 of a grain,or even less.Apart from Kassowitz's strong advocacy,the drug is well worth a trial.The phosphates are now largely used;that of lime may be given in powder (tricalcic phosphate),or in a slightly sweetened acid solution (solutions and syrups of the phosphate,or of the chlorhydro- or lactophosphate of lime.At the present day the glycerophosphates, which seem to be more directly assimilable,are often prescribed in doses of one to four teaspoonfuls a day. Phosphates are much inferior in effect to the hypophites of the Pharmacopoeia,with or without iron.

There are various other drugs utilized in rickets according to the indications presented.Thus,when anaemia is troublesome preparations of iron are required.The syrup of the iodide of iron may be given, as many drops three times a day as the baby is months old,or from ten to twenty-five drops three times a day to children of from one to three years.When the spleen is large and the lymphatic glands tumefied,three daily doses of one-half to one drop of Fowler's solution are beneficial.Fresh meat,and the juice of acid fruits, such as oranges,grapes,and lemons should be administeredwhen there is scurvy or haemorrhage from any part.

No special rules can be laid down for the treatment of the complications.The head demands special attention when it is the seat of craniotabes.The pillow ought to be soft,but not hot;no feather pillow should be allowed.The copious perspiration of the scalp requires that it should be kept cool,the perspiration wiped off frequently to avoid its condensing into water,and the flattening side of the head may be imbedded in a pillow with a corresponding depression. Copious perspiration indicates the frequent washing with vinegar and water (1 : 5-6).The muscular debility demands great caution.The infant must not be carried

on the arm,but on a pillow which supports both back and head,or in a little carriage.No sitting must be allowed until the back will no longer bend to an unusual degree.No walking must be encouraged at any time.The patient will walk of its own accord when it is time.The bones are so fragile that great care is needed sometimes not to fracture them,and to avoid periosteal pain in lifting.The child should be taken out into the fresh air as much as possible.It is desirable that the skin should undergo some training by gradually accustoming the patient to cool water.It can be readily,but gradually,reduced to 70°F. for a bath at any season;and the addition of some salt is of service at all times.

Fresh air is always a prime indication in the management of rickets;and in the same way that salt-bathing is beneficial,so is sea-air.Indeed,the latter has an admitted advantage over that of the interior; and the effects of a prolonged sojourn of,rachitic children at the seaside are often striking.Our own countryman,R.Russel,was the first to proclaim the advantages of sea-air,sea-bathing,and sea voyages (thalassotherapy),and it was at Margate that the first maritime station was established.Others on the Continent,Berck-sur-Mer,Viareggio,etc.,were established later.Some years ago,a society called "l'Oeuvre des Hôpitaux Marins" was founded in France, with the object of establishing maritime sanatoria for the treatment of rickets and scrofula.In Italy there have been added,to the marine stations,institutes for rickets - Milan,Turin,etc. - where children receive both treatment for their disease and education. The marine treatment for rickets is superior to all others,and is all-sufficient for mild cases,and for those of medium severity.After a few months' residence at the seashore,the children grow almost out of recognition - the curvatures are straightened,the anaemia disappears,and all the vital functions become re-established with energy.That which is the most striking,and which enables us to measure the rapid results of thalassotherapy,is the enormous growth in weight and in the height of the children.The best results of marine treatment are obtained in very young children who make a prolonged stay at the seaside,but we cannot expect to cure the disease by this means when the deformities are of long standing,and when the bones have become eburnated.Thalassotherapy is therefore particularly indicated at the very outset of the malady.Whenever the child is strong enough to bear them the rachitical child should have sea baths, though it often profits well enough without them. In the absence of sea baths,salt baths should be given at home,for they have a beneficent action,although an indirect one,on the general nutrition.This point has been alluded to above.The natural chlorinated sodic waters may advantageously replace these baths at home, and are to be recommended when the patient's parents can afford them.Says Foix,of Salins-de-Béarn (1883): "Rachitis in its early stages is cured constantly, and with extreme rapidity.My custom is to omit all other treatment,while the baths are being employed, but to prescribe an appropriate medical course after their discontinuance.After the third or fourth bath

the results are most evident, the curvatures of the bones have already diminished, and, repose aiding, they rapidly disappear. When the malady is more advanced the results are slower but more or less constant; the curvatures of the limbs and the swelling of the epiphyses disappear." This author looks upon the deformities of the pelvis, the vertebral column (which yield, however, to the combined use of douches and of a suitable corset), and those of the sternum and ribs as by far the most resistant to treatment, but even in them, when his treatment is adopted, takes a hopeful view of the case.

Gutteridge advocates the inhalation of oxygen for rachitical cases; and this treatment appears to be followed by some benefit.

The electrical treatment of rickets has been much vaunted by Tedeschi, of Padua; who, in 1882, introduced medication by means of continuous currents and the electric bath. In usiny continuous currents we apply the electrodes along the spinal column, one pole at the right of the cervical vertebrae, the other at the left of the lumbar vertebrae, the current being passed for two minutes in a downward, and two minutesin an upward direction; this is to be repeated every day. Tedeschi had only two failures in thirty-seven cases; and he says that soon, under the influence of this treatment, there is disappearance of the insomnia, the pains, the spasm of the glottis, and other troublesome manifestations of the disease.

Craniotabes is relieved by the general hygienic and medicinal treatment of rickets, special attention, however, being paid to the condition of the head. The general plan of treatment of this complication has been outlined above. Concentric brain symptoms require appropriate treatment; great convulsibility, the bromides, chloral hydrate, and mild opiates, which appear to be well tolerated in this condition. It is particularly in laryngismus stridulus that they are indicated in, alongside the antirachitical, hygienic, and other remedial medication. Fully developed laryngismus stridulus gives but little time for any treatment. The single attack of crowing inspiration may be cut short by shaking the infant, by slapping the face or chest with a wet cloth, or by employing the spark of a Leyden flask; for the arranging and application of the interrupted current there is hardly any time. Marshall Hall says that tracheotomy should be performed in these cases, but no one appears to have taken up his suggestion, as such procedure is scarcely justified by the condition of the patient.

Unless extreme, the deformities of the limbs that may be left after the disease has run its course require no special treatment; for they tend to disappear of themselves as the child grows older. If, however, the deformities are persistent or increasing, recourse must be had to surgical and orthopaedic measures, and especially if all possible otherwise has been done and the maritime treatment has been tried. In the case only of very young children can the curvatures in rachitical bones be reduced manually. The patient should always be anaesthetized, by means of ether or chloroform; and, in order to prevent a return of the deformity, the limbs, after straightening, should be placed in a well-padded retentive apparatus. V. Robin,

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